Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

Joint Pan American-AVMA Meeting, Kansas City, August 23-27, 1959

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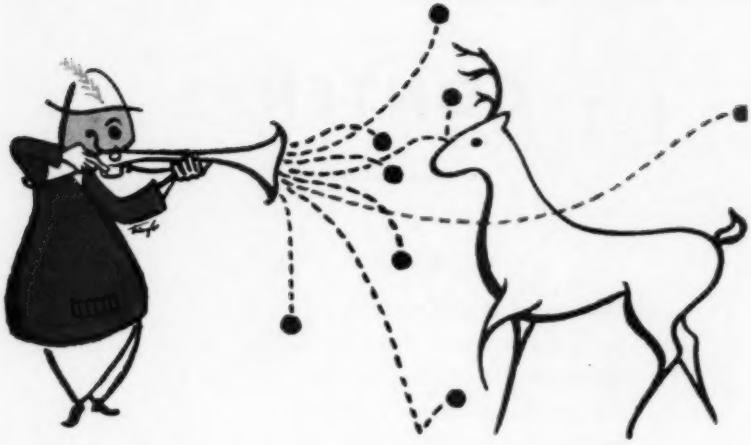

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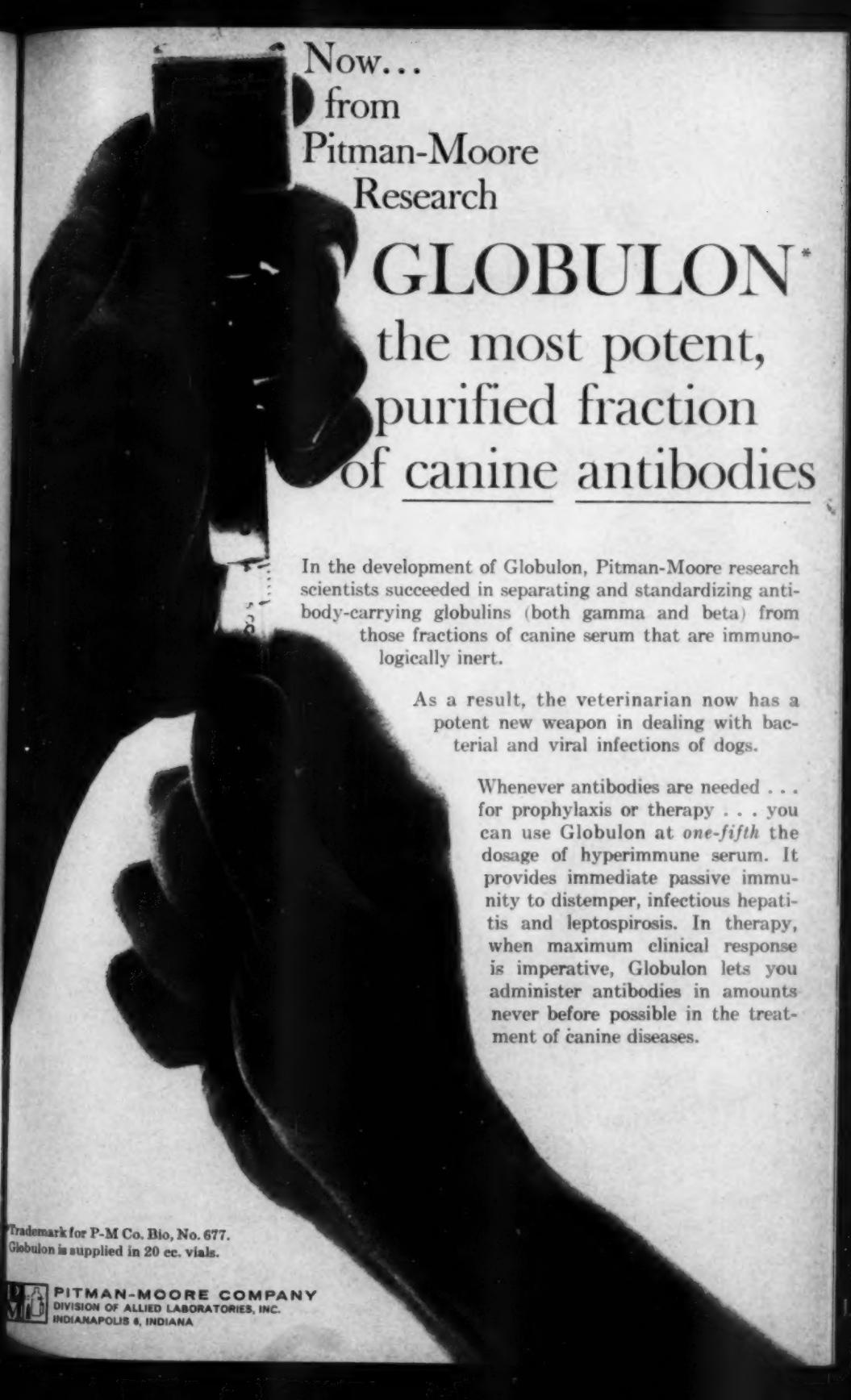

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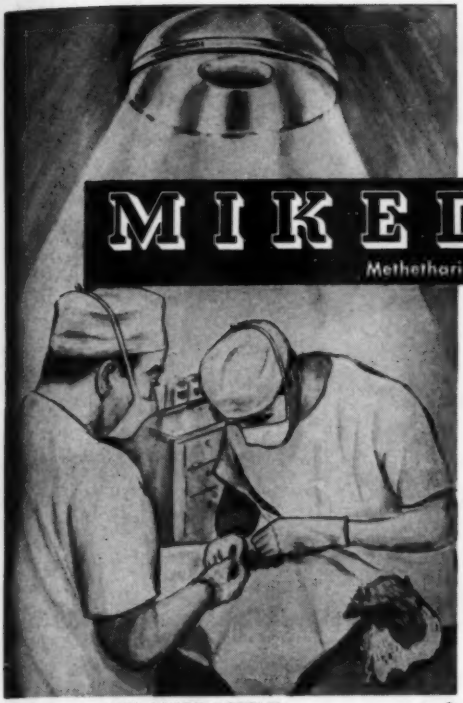

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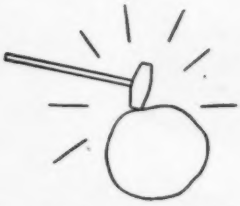

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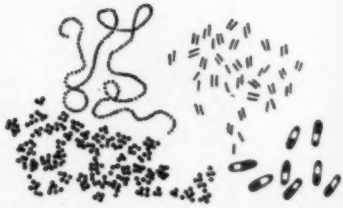

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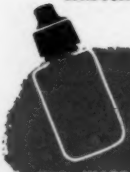

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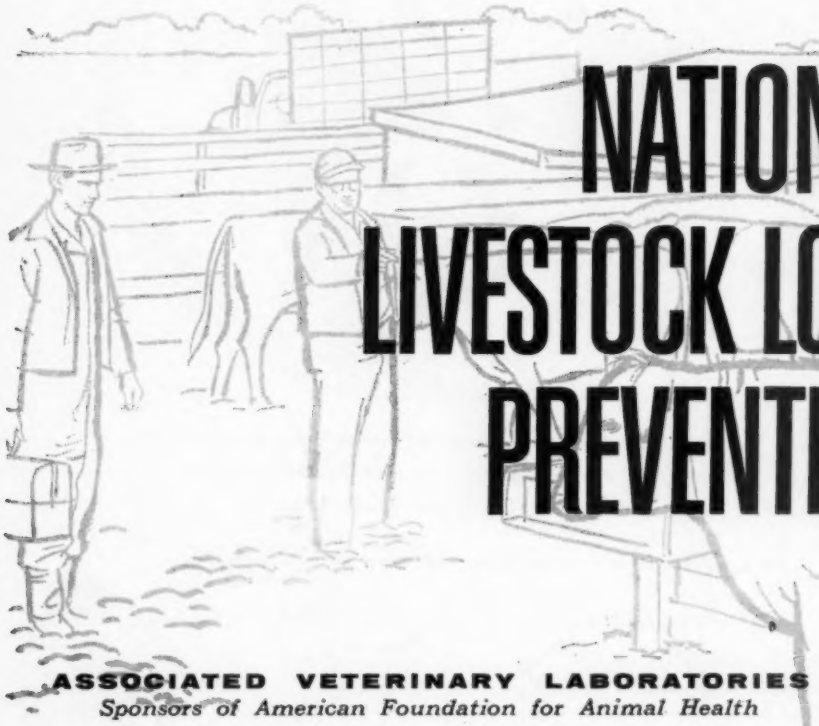

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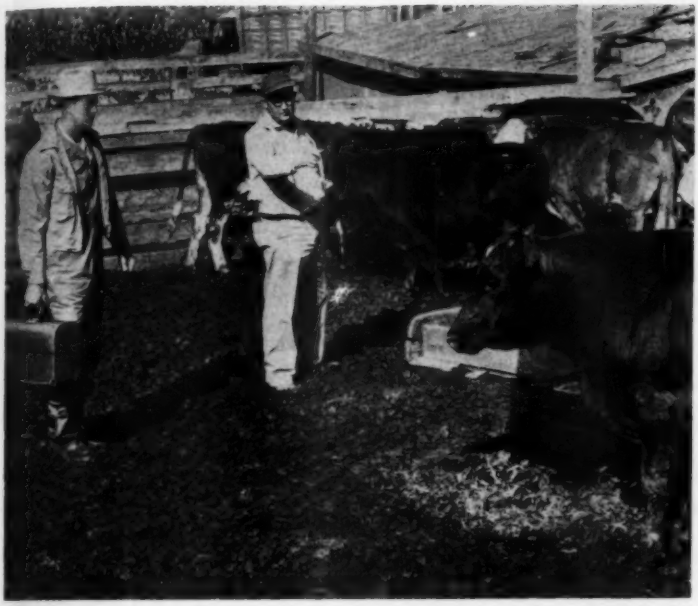

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FROM THE AVMA WASHINGTON OFFICE

U.S.D.A. Appropriations Become Law

U.S.D.A. appropriations bill for fiscal year 1960 became Public Law 86-80 when approved by the President on July 8, 1959 (see JOURNAL, Aug. 1, 1959).

Bills Passed by the Senate

The Senate passed the following bills, in which concurrence of the House was requested: S. 864, livestock and poultry disease protection on July 16, 1959 (see JOURNAL, Aug. 1, 1959, adv. p. 12 and July 15, 1959, adv. p. 12); and S. 2162, health benefits program for government employees on July 20, 1959 (see JOURNAL, Aug. 1, 1959, adv. p. 12).

P.H.S. Commissioned Corps

Senate Committee Labor and Public Welfare on July 17, reported out S. 2220, P.H.S. Commissioned Corps Personnel Act of 1959, with amendments (see JOURNAL, Aug. 1, 1959, adv. p. 14).

Senate Hearings on Tax Deferment Bills

Senate Finance Committee resumed hearings July 15 on tax deferment bills (see JOURNAL, July 15, 1959, adv. p. 12). Dr. J. O. Knowles made a strong case for enactment of H.R. 10, in behalf of AVMA, both in direct testimony and during interrogation by committee members. Sen. Bennett, when questioning a subsequent witness, commented favorably on answers given by the AVMA witness. Hearings recessed subject to call of Sen. Byrd, chairman.

Proposed Amendments to Social Security Act

H.R. 7952, Rep. Mills (D., Ark.), to effect recommendations on Social Security Financing with respect to investment of F.O.A.I. Trust Fund and Federal Disability Insurance Trust Fund. H.R. 8099, Rep. Fogarty (D., R.I.), and H.R. 8108, Rep. Rodino (D., N.J.), to remove limitations on outside income one may earn while receiving benefits under the Act. H.R. 8147, Rep. Osmers (R., N.J.), to increase amount of earnings permitted without suffering deductions from $1,200 to $1,800. H.R. 8263, Rep. (Mrs.) Dwyer (R., N.J.), would permit $2,400 outside earnings (or $3,600 for widow with minor children).

School Lunch Act

H.R. 8104, Rep. Johnson (D., Wis.), would transfer administration of National School Lunch Act from Secretary of Agriculture to Secretary of Health, Education, and Welfare.

Agricultural Policy

S. 2395, Sen. Stennis (D., Miss.), would establish a commission on agricultural policy.

Health Benefits Program, Federal Employees

H.R. 8210, H.R. 8211, H.R. 8212, by Representatives Morrison (D., La.), Porter (D., Ore.), and Davis (D., Ga.), to provide a health benefits program for government employees. Similar to but not identical with S. 2162 (see JOURNAL, Aug. 1, 1959, adv. p. 12).

Annual Leave, Federal Employees

S. 2325, Sen. Sparkman (D., Ala.), would provide that any annual leave in excess of maximum amount which can be carried into next leave year, shall be credited to employee's sick leave account.

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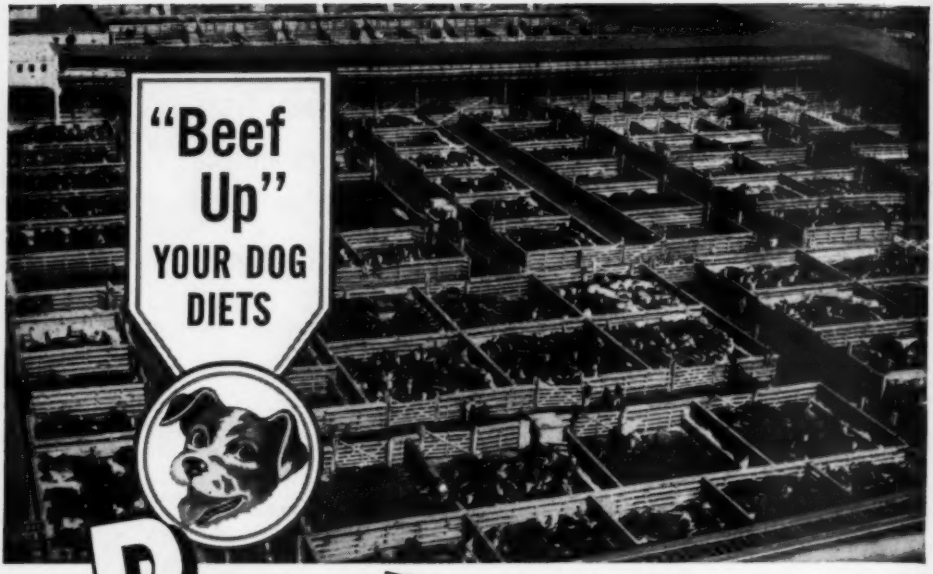

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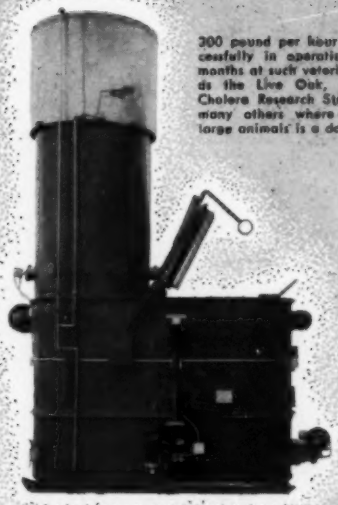

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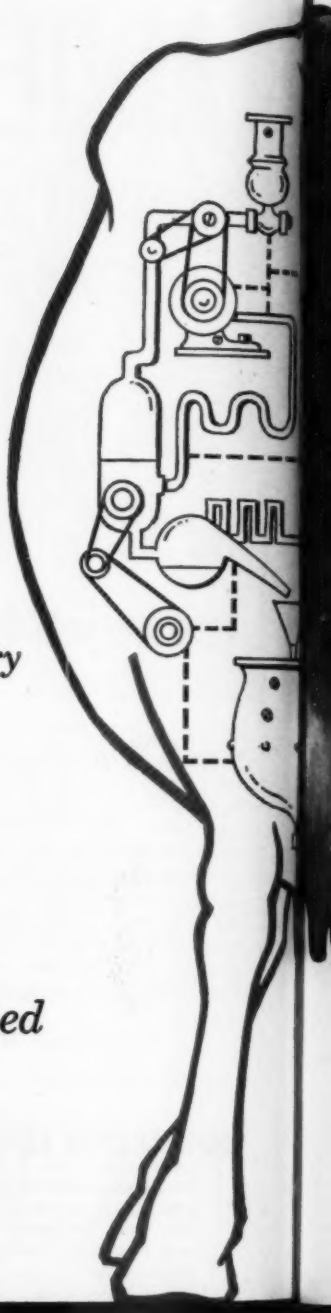

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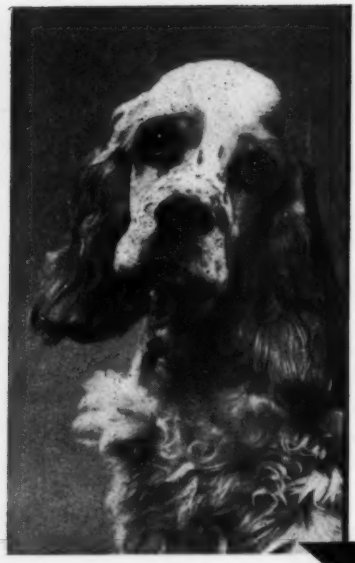

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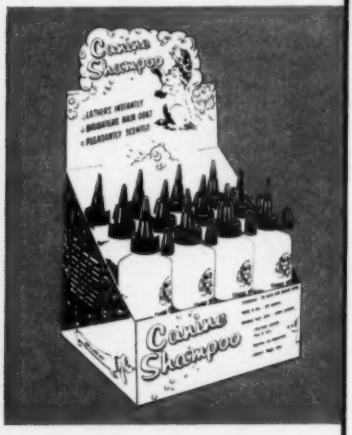

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The Genetic Aspects of Hip Dysplasia in Dogs

J. G. SNAVELY, M.D.

Toledo, Ohio

DYSPLASIA OF THE hip is not an uncommon disease of man and most breeds of dogs, and there is a remarkable similarity in the clinical, radiological, and necropsy findings in both species. For example, one monograph[4] on dysplasia of the hip in man could serve equally well for dogs except for the section on therapy.

Just as there is a superficial similarity in many features of the disease in either species, there is a similarity in the variance of opinions as to its causation. Several authors[1-3] believe the disease in man to be genetically transmitted, but another[5] states, "There are at least three possibilities: (1) It may be hereditary; (2) it may be due to a local vascular or metabolic disturbance in early embryonic life; and (3) it may take origin from some intrauterine mechanical force in fetal life." Some breeders of dogs consider dysplasia genetically conditioned and one[6,9] has recently reported two studies which support this view. Others are of the opinion that factors of diet, hypervitaminosis or vitamin deficiency, trauma, or a host of other factors might be responsible for the disease.

While similarities of the disease do not establish identity of the condition in two different species, the possibility of studying the genetic factor in dogs offers considerable speculative interest. The study reported here was begun to eliminate the genetic factor, if possible, in order to consider the nongenetic possibilities.

Dr. Snavely is director of Laboratories and Medical Education, Maumee Valley Hospital, Toledo, Ohio.

Presented before the Section on Small Animals, Ninety-Fourth Annual Meeting, American Veterinary Medical Association, Cleveland, Ohio, Aug. 19-22, 1957.

DEFINITION OF TERMS

The general term "hip dysplasia" (as used here) means a failure of proper development of the hip joint as a whole, manifested radiologically by varying degrees of failure of proper fit of the head of the femur to the acetabulum; clinically, by varying degrees of abnormality of gait; and at necropsy, by varying degrees of reaction of the soft and bony tissues to the constant micro-trauma implicit in the abnormal mobility of the femoral head. The term "subluxation of the hip" is used to describe the result of dysplasia when it is sufficiently severe to permit the femoral head to move in and out of the acetabulum. Subluxation, then, is merely a manifestation of the degree of severity of dysplasia and, as such, is a descriptive, not a diagnostic, term.

The term "taint" has been used to imply minimal lesions. This term is not used here.

"Aseptic necrosis" or "Legg-Perthes" changes are used to define radiological changes which cannot be assumed necessarily to be due to dysplasia, and cannot be used as synonymous with dysplasia but may result from it.

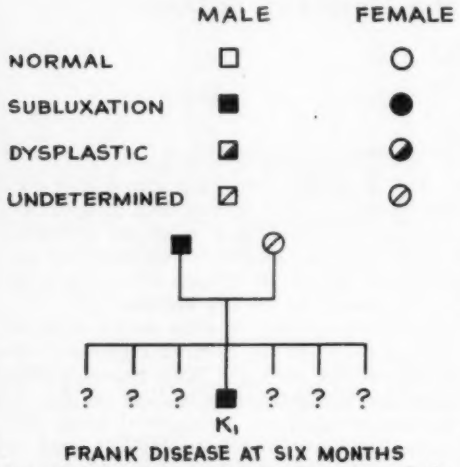
CRITERIA OF DIAGNOSIS

The radiological criteria of this disease in dogs have been set forth[10] and are used throughout this study.* All films were read completely independently by the author and two others.** Using uniform criteria, no significant variance of interpretation was found. However, in the course of the study, certain films selected at random were submitted to various centers and widely variable diagnoses were obtained,

ranging from normal to frankly abnormal. The internal consistency in the study is the effect on a single set of criteria, and cannot be considered as evidence for their general acceptability.

The clinical criteria of the disease are legion and should be divided into two groups.

1) Clinical signs indicating the possibili-



FRANK DISEASE AT SIX MONTHS

Fig. 1—Genetic chart showing the origin of the dysplastic male used as the sire for subsequent dysplastic breedings.

ty of dysplasia but requiring a radiograph for confirmation are:

(*a*) excessive awkwardness in pups with respect to gait, particularly after periods of rest or intense exercise; (*b*) hitching or "cog-wheel" gait; (*c*) front leg lameness; (*d*) visibly broadened hips; (*e*) waddling gait with a tendency to fall frequently to one side; (*f*) poor exercise tolerance; (*g*) awkward, asymmetrical sitting position; (*h*) tendency to sleep on back; (*i*) asymmetric hips.

2) Diagnostic signs, sufficient in themselves for diagnosis.

There are only two such diagnostic signs. The first of these is the square croup usually associated with a "cog-wheel" gait. This sign is present only in advanced dysplasia with subluxation and is pathognomonic. The second of these is Ortolani's[**] sign ("sign of the jerk"). The "jerk" or "click" is due to passage of the femoral head in or out of the acetabulum. It ranges in degree from a palpable impulse over the greater trochanter to an audible click which can be heard several feet away. It may not be present in simple dysplasia or in advanced cases where the acetabulum is totally destroyed. When present, it is a characteristic and reliable sign of the disease. It is best produced by passive extension of the leg or by rocking the hips with the dog standing with the back legs extended.

NECROPSY CRITERIA

The necropsy criteria of the disease are products of both age and the degree of abnormal mobility. In younger dogs and pups, these consist of:

1) Increase in the amount of joint fluid. In normal pups, the amount of fluid is sufficient to moisten the articular surfaces. In dysplastic pups and young dogs, gelatinous fluid under slight pressure is a constant finding. Aspiration of normal joints yields the maximum of a drop of fluid; abnormal joints yield 0.5 ml. or more.

2) An abnormally shaped femoral head is regularly found. The head is shortened in its long axis and is not hemispheric. This change is minimal and frequently difficult to see.

3) The joint capsule is grossly thickened, and microscopic sections show chronic papillary synovitis in varying degrees of severity.

4) There is elongation and softening of the ligamentum teres. In older dogs (1 year or more), erosion of the acetabulum, marked thickening of the joint capsule, adventitious calcification of the periarticular fibrous tissue, extreme flattening of the femoral head, loss of ligamentum teres, and severe papillary synovitis make the necropsy diagnosis quite simple in comparison. Even in mild dysplasia, older dogs show easily demonstrable papillary syno-

*The following criteria were used in this study to substantiate the x-ray diagnosis of dysplasia: (1) shallow-set, nonspherical acetabulae (either shallow in concavity or deep half-lemon shaped); (2) failure of appropriate fit of femoral head to acetabulum; (3) frank displacement of the femoral head; (4) secondary changes of the acetabulum consisting of erosion of the margins of the acetabulum; (5) calcific lipping or spurring of the acetabular rim; (6) periarticular calcification; (7) channelization sign; and (8) secondary distortion and erosion of the femoral head.

Numbers 5, 6, and 8 are always in conjunction with other obvious findings.

In most instances, three radiographs were taken of each animal: one with the animal on its back and the legs extended, the second view with the same position but the toes sharply turned in, and the third view with the same position with the toes sharply turned outward. Anesthesia was used only with a small number of the animals early in the study. This was later found not to be necessary.

**E. J. Dranginis, M.D., Toledo, Ohio, and Andrew Wheeler, D.V.M., Okemos, Mich.

vitis to varying degrees. The necropsy findings in young dogs are minimal and easily missed. The necropsy criteria are not as reliable as radiological methods.

To summarize the use of the above criteria and definitions, the clinical impression was primarily used to determine the most advantageous time to take radiographs; the radiograph was used for the primary diagnosis. Complete necropsies were done primarily to assay the value of such examinations with reference to the hips and to record other associated lesions. Two pups died too early to be of diagnostic significance and were excluded from this study. One had congenital heart disease (interventricular septal defect); the other had intussusception of the small intestine, with gangrene.

EXPERIMENTAL CONDITIONS AND METHODS

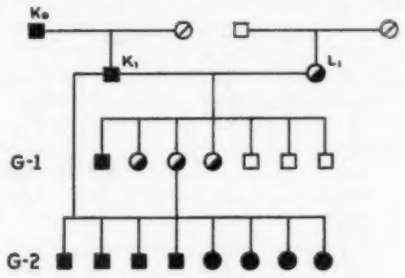
The experimental animals consisted solely of purebred German Shepherd Dogs. Adults were fed commercial ration of various sources and meat supplement. In addition, bitches in whelp and pups were given reconstituted powdered milk and cod-liver oil. Fat was added to give roughly 20 per cent fat in the diet of pups and 10 per cent in that of adults. All adults and pups over 10 weeks old were actively immunized against distemper and hepatitis. All except nursing pups and bitches were kept under outdoor conditions. Reasonable freedom from parasitic infection was maintained by appropriate anthelmintics. All animals were maintained in excellent health and in good coat.

Pups were observed regularly after 3 weeks of age for clinical evidence of dysplasia, and radiographs were taken at less than 3 months of age only if clinical evidence of disease was present. Radiographs at 3 months, 6 months, and 1 year were taken irrespective of presence or absence of clinical signs.

In the genetic charts (fig. 1-5), differentiation is made between mild and severe lesions. This is not intended to infer any basic differences in diagnosis but is simply descriptive. This differentiation is for tabular purposes only. Statistically, no such differentiation is made. Any lesion, mild or severe, is considered abnormal.

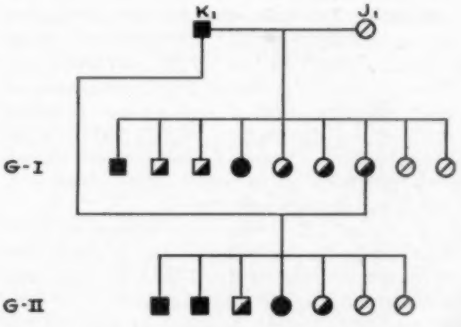
RESULTS

The origin of the dysplastic male used as the sire for subsequent dysplastic breedings is illustrated (fig. 1). The dysplastic male, designated K_1, initially had signs of a left front leg lameness at 6 months. Radiographs at 10 months revealed advanced dysplasia of the left hip with frank subluxation. The right hip was also involved but to a much less-marked degree. The sire (K_0) of this dog, on subsequent investigation, was determined to be dysplastic by x-ray studies. The dam was not examined radiographically but clinically showed no evidence of dysplasia. The condition of the



G-2 DISEASE EVIDENT AT 10 WEEKS CLINICALLY AND BY X-RAY

Fig 2—Results of the preliminary breeding of this dysplastic sire (K_1—see fig. 1) to a dysplastic female (L_1); the sire (K_1) was then bred to one of his dysplastic daughters.



BOTH GENERATIONS DYSPLASTIC (X-RAY) AT 3 MONTHS — TWO MALES IN G-II CLINICALLY APPARENT AT 12 WEEKS

Fig. 3—Results of a second dysplastic breeding using the same sire (K_1) and a female (J_1) whose x-ray findings were unknown but whose siblings included several dysplastic animals; the sire (K_1) was then bred to one of his dysplastic daughters.

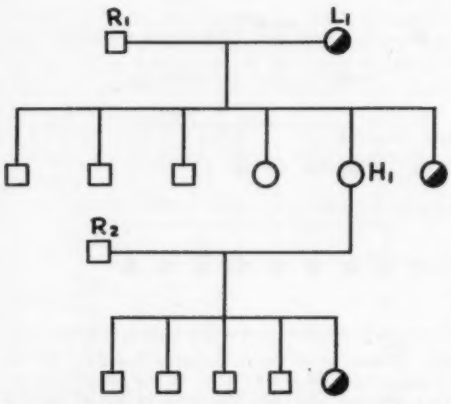


Fig. 4—Results of the breeding of a normal male (R_1) to a dysplastic female without subluxation; a normal female of this generation was then bred to a second normal sire (R_2).

siblings of K_0 and K_1 is unknown to the author.

The result of the preliminary breeding of this dysplastic animal (K_1) to a dysplastic but not subluxated female (L_1) was charted (fig. 2). The dam (L_1) was selected because her sire was known to be radiologically normal and her dam, although not available for radiographic examination, was clinically normal. There were 7 pups in the litter, all of which survived. Of these, 1 male developed front leg lameness at 8 months, and a radiograph revealed unilateral dysplasia with subluxation (right side). Three females were shown radiographically to have mild dysplasia, but 3 males were not.

The sire (K_1) was then rebred to 1 of his dysplastic daughters and 8 pups survived of a litter of 10. The 4 females and 4 males all showed varying degrees of dysplasia, and clinical evidence of subluxation was present in all. A striking finding was the early development of noticeable clinical signs. Half the pups showed frank clinical evidence of dysplasia by 10 weeks of age.

A second dysplastic breeding (fig. 3) used the same sire (K_1) with a female (J_1) whose x-ray findings were unknown but whose siblings included several dysplastic animals. The first breeding produced 9 pups but the progress of 2 could not be followed. Of the 7 remaining, all had dysplasia, 2 with subluxation and the remaining 5 showed mild but definite x-ray evidence of the disease. Those showing mild dysplasia did not develop clinical evidence of the disease at maturity.

The sire (K_1) was then rebred to a dysplastic daughter of this mating. There were 7 offspring, the progress of 2 of which could not be followed. Of the 5 remaining, 3 showed frank evidence of dysplasia in severe form; 2, in mild degree. Both males in the second generation were clinically affected at 12 weeks.

A normal male (R_1) was bred to a dysplastic female without subluxation (fig. 4) identical to L_1 in figure 2 of the dysplasia series. R_1 was 1½ years old at the time of the first breeding. He was examined by three different persons, in widely separated parts of the country, at 3 months, 1 year, and again at 2 years of age. The initial breeding yielded 6 pups of which 5 had neither clinical nor x-ray evidence of dysplasia, but the sixth demonstrated mild but definite dysplasia of one hip and was killed.

A normal female of this generation was bred to a second normal sire (R_2), of which

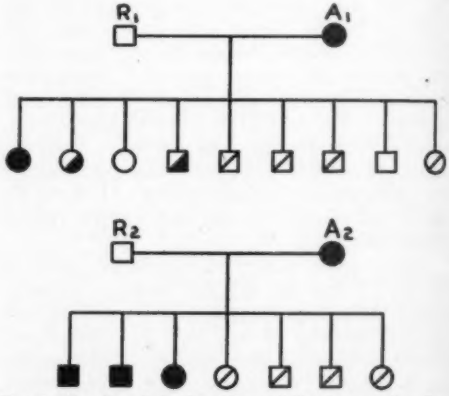


Fig. 5—Results of the breeding of the 2 normal males (R_1 and R_2—see fig. 4) to females with subluxation.

mating 5 pups resulted. The hips of 4 male pups were completely normal but 1 was monorchid. The fifth pup, a female, had moderately advanced dysplasia and was killed at 7 months.

Each of the normal males was bred to a female with subluxation. The R_1-A_1 mating (fig. 5) produced 9 pups, of which 5 were available for follow-up studies. Of these, 3 had dysplasia. The second male (R_2) was

bred to a frankly dysplastic female (A_2), but of the 7 resulting pups, only 3 were available for follow-up studies. All 3 were severely dysplastic.

A tabulated analysis (table 1) was prepared to show the incidence of the disease under the conditions of purposeful breeding of dysplastic parents. The disease was actually diagnosed in 87 per cent of the animals. At N=30 and P=0.01, the population limits of this incidence are 63.6 to 97.7 per cent.

A summary was prepared (table 2) to show the incidence of dysplasia in the series using 2 normal males (fig. 4, 5). At N=14 and P=0.01, the population limits of this percentage are 2.6 to 59.0 per cent.

This would indicate a significant difference in the two populations greater than that due to chance at a high level of significance, and we attribute this difference to the eminence of genetic factors which condition the development of the disease.

This statistical analysis was done when the projected experimentation was incomplete. Such marked differences were not anticipated but there appeared to be no reason to carry on further investigation when significant differences could be shown in spite of the small numbers. The statistical parameters are derived from binomial confidence limits tables.[6]

The purpose of the experiment was to eliminate, if possible, genetic factors. Obviously, this could not be done and, in fact, quite the opposite was proved. The defect in number of animals is largely in the so-called "normal" group.

Subsequent to the completion of these data, three additional pairs of normal parents have been bred. Eighteen pups resulted, 3 of which showed dysplasia. This incidence is 17 per cent, thus falling closely to the percentage of change in previous breedings. If these are added to the data given in table 2, the over-all percentage of dysplasia is 19 per cent. The population limits for the whole group would be 5.1 to 42 per cent incidence. The gap between the two populations is thus even greater with the larger number of animals and equal numbers in each group.

ANCILLARY INFORMATION

During the period of the experiment, many dogs from nonexperimental breedings but mostly German Shepherd Dogs were submitted to the author for clinical, x-ray, or necropsy investigation. Interestingly, these dogs frequently showed more severe and earlier manifestations of dysplasia than the experimental pups from dysplastic parents. Obviously, the selection of these was biased since animals seen to be partially crippled by untrained observers, without the advantage of x-ray facilities, would necessarily be the more severely affected.

TABLE 1—An Analysis of the Incidence of Dysplasia in Dogs Resulting from Purposeful Breeding of Dysplastic Parents

Disease present in population	57
Disease absent in population	4
Total	31
Percentage disease present	87%
At N = 30, population limits for P= 0.01 are 63.6 to 97.7%	

Certain breeders, however, submitted breeding stock in which clinical findings were absent and a high level of incidence of dysplasia was present. In one instance, of 9 dogs of imported breeding, there was only 1 normal dog. In another instance, of 8 dogs of American breeding, only 2 could be considered normal.

These observations suggest the wide diffusion of the dysplasia trait in German Shepherd Dogs. Unfortunately, similar studies of other breeds have not been carried out, and the findings, while strongly suggestive, do not eliminate the factor of chance.

TABLE 2—An Analysis of the Incidence of Dysplasia in Dogs Resulting from Purposeful breeding of Non-Dysplastic Parents

Disease present in population	3
Disease absent in population	11
Total	14
Percentage disease present	21%
At N = 14, population limits for P = 0.01 are 2.6 to 59%	

DISCUSSION

In statistical consideration of the experimental findings, no attempt is made to distinguish between mild or severe disease or whether the lesion is unilateral or bilateral. Actually, in many instances where apparent unilateral involvement was present, careful examination showed the opposite joint also to be affected.

Insufficient numbers of animals were present in the experimental groups to make any comments with respect to the relation of sex to development of the disease. The ancillary experience with dogs of random

breeding, however, strongly suggests that females tend to be more frequently affected than males and that their disease is generally more severe and more frequently obviously bilateral.

With respect to the genetic basis of the disease, we feel that we have demonstrated that the disease is genetically conditioned. We are of the opinion that the number of animals and breedings reported here is too small to draw any conclusions as to the precise nature of the genetic component. The number of animals is also too small to exclude other factors which might influence the penetrance or expressivity of dysplasia. The regularity of transmittance of the disease from parent to offspring suggests the trait to be dominant, and this view is supported by the transmission of the disease to offspring from one involved parent. However, far larger numbers of experimental breedings are needed if details of the type of genetic factor are to be determined accurately. Further investigation to determine the precise genetic mechanism is important, for this will bear on the possibility of eliminating the fault.

With respect to elimination of the disease trait, the relatively high incidence of dysplastic pups from radiologically normal parents should be recognized. It is interesting that in genealogical charts,[2] similar situations in man are not uncommon. In these instances, afflicted siblings or ancestors of one parent were regularly found. Keeping in mind the apparent wide diffusion of the trait in the German Shepherd Dog and the relatively high incidence of dysplastic pups from normal parents, the statement that ". . . it should be a relatively easy matter to reduce the incidence of dysplasia of the hip in dogs to the point at which it becomes a rare disease"[3] is at the least an oversimplification of the problem.

Certain of the animals of the experimental group were permitted to survive up to 4 years of age (1957). Although the lesion is inexorably progressive radiologically, the clinical manifestations appear to stabilize at an early age (about 1 to 2 years). Frequently, the gait is so smooth and flowing that it is difficult to believe that the x-ray changes and the absence of clinical signs are compatible. The absence of clinical criteria is then inconclusive as to the presence or absence of the disease or the suitability of the animal for breeding purposes.

SUMMARY

1) Evidence is presented to show that dysplasia of the hips of dogs is significantly increased by purposeful breeding of dysplastic parents.

2) It is assumed that this increase is genetically conditioned.

3) The number of animals is too small to elucidate the mechanism of transmission of the defect.

ADDITIONAL OBSERVATIONS

Further observations have been made since the completion of the original study in August, 1957. No further breedings have been made of animals where both parents were dysplastic; however, six matings were made in which 1 parent (the female in each case) was dysplastic. This resulted in 42 pups which lived to an age of 8 months or more. Of these animals, 62 per cent showed evidence of dysplasia by the same criteria used in the original study.

Five breedings were made between normal parents. Thirty pups living to 8 months of age or more resulted. Of these, 83 per cent were radiologically normal by the criteria outlined.

These findings appear to substantiate in every detail the predictions made from the original study and also strongly confirm the wide diffusion of the dysplasia trait in German Shepherd Dogs and a significant rate of the lack of penetrance. Further studies in the understanding of the genetic background require the development of homozygous normals with respect to the trait.

It is interesting and perhaps of great significance that of all of the breedings controlled by this writer, there resulted only one litter of 6 in which each of the offspring could be demonstrated to be normal radiologically.

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Hereditary Acetabular Dysplasia in German Shepherd Dogs

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ALTHOUGH congenital subluxation of the hip in man has been recognized for centuries, it was first described in the dog in 1935.[8] It has been suggested that the defect is most common in the German Shepherd Dog[8] and many consider it to be hereditary.[1,2,4,6,7] Various theories have been advanced to explain the mode of inheritance. Uncertainty over the precise nature of the lesion is reflected in the variety of names used to designate the defect in both man and dogs. Names such as "congenital luxation" (dislocation) of the hip, "hereditary subluxation," "hip dysplasia," and "acetabular dysplasia" have all been used. The disease is often not clearly defined and, in the case of dogs, it has often been confused with Legg-Perthes' disease.

A detailed analysis of the incidence of the lesion among the various breeds of dogs has not been published. Most observations, however, have been made on German Shepherd Dogs. A systematic investigation of the disease in this breed has been carried out in Sweden since 1956, and the purpose of this paper is to present the results which have been obtained.

The ultimate goal of this investigation is to answer the following questions: (1) How should this disease be defined? (2) What is the incidence of the disease among German Shepherd Dogs in Sweden? (3) Is the disease hereditary and, if so, what is the mode of inheritance? (4) What is the practical clinical significance of the lesion?

MATERIALS, METHODS, AND RESULTS

1) A total of 471 German Shepherd Dogs, some clinically healthy and others with hindlimb lameness, was radiographed in the manner described.[5] Of these animals, 295 presented a uniform appearance. In ventrodorsal symmetric projection with the hindlimbs flexed and abducted to the maximum degree, the acetabula of these dogs appeared as a half-circle and occasionally somewhat deeper. The acetabula of the other 176 dogs deviated from this normal appearance. Various degrees of shallowness of the acetabula were found in this group. There is no definite correlation between the degree of acetabular dysplasia and the occurrence of subluxation or luxation.[5] Secondary changes, such as spur formations on the edge of the acetabulum and head of the femur, apparently occur only as sequelae to subluxation. In 138 dogs (78.4 % $\pm$ 3.1), the lesions were bilateral. The essential feature in all instances was a varying degree of unilateral or bilateral acetabular shallowness. Traumatic hip dislocations with normal acetabula should be clearly distinguished from the disease described here. We have observed a few instances of a tendency toward subluxation in conjunction with normal acetabula. Dogs with this tendency have been excluded from our present material, but are subjects for further study.

2) Roentgenograms were made, in the manner described, of all available German Shepherd Dogs belonging to the Swedish Armed Forces. This random group of 191 dogs represented almost all the significant breeding lines in Sweden. The clinical status of these dogs was ignored for the purposes of this study. According to the criteria established above, 51 of these dogs (26.7% $\pm$ 3.2) showed signs of acetabular dysplasia. These dogs are included in the 471 described in the preceding para-

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graph and in those reported in the following paragraph.

3) Genetic analysis of dogs belonging to the Swedish Armed Forces Dog Training Center included those litters born during 1956, 1957, and the first four months of 1958 for which both parents could be examined for the defect. The pups and their parents were examined radiologically. It was apparent (table 1) that there is a genetic background for the occurrence of the defect. Matings in which 1 or both animals were defective resulted in a significantly greater proportion of defective pups than did matings between normal animals.

TABLE 1—Preliminary Genetic Analysis of 191 German Shepherd Dogs

Mating	Offspring ♂		♀		♂+♀*		D-frequency		
	N	D	N	D	N	D	♂	♀	♂+♀*
N ♂ x N ♀	42	4	24	10	66	14	0.087	0.294	0.175
N ♂ x D ♀	22	13	31	8	53	21	0.371	0.205	0.284
D ♂ x N ♀	5	3	9	7	14	10	0.375	0.438	0.417
D ♂ x D ♀	4	5	3	1	7	6	0.556	0.250	0.462
Total	73	25	67	26	140	51	0.255	0.280	0.267

♂ = male, ♀ = female; N = normal (free from dysplasia), D = with dysplasia; * $x^2 = 8.83$; $f = 3$; $0.05 > P > 0.01$.

Some writers,[2] who define the defect as a subluxation, maintain that inheritance is regular, monogenic, and recessive. Another considers the defect, defined as dysplasia of the hip joint, to be monogenic and dominant.[6,7] On the basis of our investigations, however, it appears that the defect is not inherited as a regular, monogenic characteristic.

Matings between defective parents produce normal as well as defective offspring, a result which is incompatible with a regular, recessive inheritance. A monogenic, dominant mode of inheritance is also unlikely since matings between normal animals yield a certain proportion of defective pups. With our definition of the defect, incomplete penetrance of a possibly monogenic characteristic must be assumed.

The incomplete penetrance may reflect either the effects of external environment or the effects of other genes upon the basic characteristic (genetic environment), alone or in conjunction.

Another aspect of interest is shown in the table. The incidence of the defect varies greatly between the sexes and, in the case of matings between normal parents, this difference is statistically significant ($0.05 > P > 0.01$). The mode of inheritance is in some manner associated with the sex of the dog. Investigations of this observation and the question of penetrance are being continued with test matings.

To estimate the actual significance of acetabular dysplasia as a cause of lameness in German Shepherd Dogs, the clinical records of all dogs of this breed admitted because of hindlimb lameness to the surgical clinic, Royal Veterinary College, during 1957 and 1958 were examined. On the basis of these records, the 85 dogs involved can be allotted to the following categories:

- 27 with changes in the stifle joint (rupture of the cruciate ligaments and osteoarthritis);
- 15 with fractures of the pelvis and hindlegs;
- 14 with acetabular dysplasia;
- 12 with spinal cord and nerve root changes, traumatic and atraumatic (disk protrusions, metaplastic bone tissue in the dura, degenerative cord changes, fractures, etc.);
- 9 with traumatic lesions of the hindlegs other than fractures (wounds and dislocations);
- 4 without diagnosis;
- 2 with muscular pain;
- 1 with osteosarcoma;
- 1 with subluxation of one hip joint without demonstrable dysplasia of the acetabulum.

DISCUSSION

Judged by the criteria we have established, about one fourth of the German Shepherd Dogs in a representative Swedish sample show lesions of acetabular dysplasia. This incidence is remarkably great. Only a few of these dogs displayed clinical signs which could be ascribed to the defect.

The incidence of acetabular dysplasia in the adult dog population is probably somewhat lower than 26.7 per cent since dogs with pronounced hip defects and lameness would probably be destroyed rather than sold. In the adult population of German Shepherd Dogs, it is thought that about 20 per cent have some degree of acetabular dysplasia.

An impression of the proportion of dogs with acetabular dysplasia, which become so lame that the owners seek veterinary advice, can be gained from the records of the surgical clinic of the Royal Veterinary College. During the years 1957 and 1958, approximately 750 German Shepherd Dogs

were admitted to the surgical clinic. It may be assumed then that 20 per cent (150 dogs) had acetabular dysplasia although only 14 showed obvious signs of lameness due to this condition.

It appears that subluxation or luxation rather than acetabular dysplasia *per se* produces clinical signs. Well-developed muscles and ligaments seem to be able to compensate to a significant degree for poorly developed acetabula, a factor which will be discussed in more detail. It is impossible to ascertain precisely the extent to which the durability of a dog is affected by the defect. There probably is a greater incidence of osteoarthritis in the hip joints of aged dogs with acetabular dysplasia than among aged dogs with normal acetabula. The usefulness of a defective dog, especially for jumping and draught, is undoubtedly reduced. For example, a dog which enjoyed success in field trials could not be entered in jumping competitions because of his poor acetabula. Many dogs with acetabular dysplasia have an unsatisfactory manner of moving the hindlimbs. However, we wish to emphasize that the conformation and gait of a dog does not necessarily reflect the state of the acetabula. A dog with bilateral total luxation, for example, was given a certificate of approval at a Swedish dog show.

The term "hereditary" seems fully justified in this defect. Of the names in current use, "hip dysplasia," "acetabular dysplasia," and "subluxation" or "luxation" (dislocation) of the hip, we believe that only the first two are adequate since the defect is not necessarily associated with dislocation. Whether hip dysplasia or acetabular dysplasia is to be preferred depends upon the essential nature of the defect. In this connection it is of interest to cite the investigators[10] who, in their attempts to determine the pathogenesis of the lesion in man, succeeded in experimentally producing acetabular changes in young pups by dislocating the femur.

"Concepts of the etiology of congenital dislocation of the hip have been evolved by a process of deduction based upon normal embryological development of the hip joint and findings in infants following hip dislocation. Numerous roles have been ascribed to the associated features of congenital dislocation of the hip, chief of which are acetabular dysplasia and anteversion of the neck of the femur. Acetabular dysplasia is regarded by some as a primary cause of congenital dislocation of the hip. Furthermore, some assign a genetic etiology to dysplasia. Others suggest that the associated features occur solely as a result of the dislocation. When based upon clinical observations alone, the argument as to which features are primary and which are secondary could be endless. The problem was therefore brought to the laboratory. . . . There is reported a high incidence of acetabular dislocation of the hip in German Shepherd Dogs. While this is an interesting observation, the situation is analogous to that in human patients, in whom our knowledge of the condition is based upon findings after the dislocation has taken place."[10]

The results of our investigation suggest that this presentation of the pathogenesis in dogs does not agree with known facts. A shallow acetabulum without radiographic signs of dislocation of the hip is, in reality, not uncommon among German Shepherd Dogs. This implies that the acetabular lesion is primary in most instances, and changes in the ligaments are secondary to this. This by no means eliminates the possibility that primary ligament changes can contribute to the occurrence of dislocation in some instances. In fact, this may explain at least in part why dogs with the same degree of acetabular shallowness often present widely varying degrees of luxation. Another explanation, of course, may be that a well-raised and properly exercised dog may be able to compensate for shallow acetabula better than a poorly managed dog. Our observations have not eliminated the possibility that ligaments as well as the acetabulum might be underdeveloped. It appears unlikely, however, that acetabular dysplasia should be only a sequela to luxation or subluxation caused by an insufficient ligament and joint capsule.

One occasionally encounters dogs with atraumatic dislocation of the hip but with normal acetabula. It is doubtful whether such dogs should be considered as displaying the syndrome described in this paper. For the present, at least, we prefer to limit our material to dogs with acetabular changes. Only 4 dogs, 2 of which were not German Shepherd Dogs, have been seen with subluxation without demonstrable trauma or acetabular dysplasia.

With our present knowledge, the term "hereditary acetabular dysplasia" seems

to be most preferable to indicate the disease described in this paper. Hypoplasia is perhaps even more suitable but would unnecessarily complicate the already luxuriant nomenclature.

Studies of the etiology and pathogenesis of acetabular dysplasia are under way in the Department of Radiology, Royal Veterinary College.

SUMMARY

Acetabular dysplasia in German Shepherd Dogs in defined as a condition in which the acetabulum in a ventrodorsal symmetric projection is reproduced on a radiograph as less than a half-circle. In 176 dogs with acetabular dysplasia the disease was found to be bilateral in 78.4 per cent. In a random sample of 191 dogs, a defect frequency of 26.7 per cent was found. Of 85 German Shepherd Dogs with lameness of the hindlegs, acetabular dysplasia was responsible for 14 cases of lameness. There is statistically significant evidence of the hereditary nature of the disease. The complex hereditary pattern is the subject of further studies. An interesting sex linkage is noticed. The etiology of the disease is discussed, and of the different names in use, "hereditary acetabular dysplasia" seems to be most suitable.

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Fluoride Concentration in Bones

After necropsy, bone analyses were made on adults who had lived for many years in an area where the fluoride content of water varied. The average fluoride level in the ribs was 500 p.p.m. for those who had been drinking water with 0.1 to 0.4 p.p.m. of fluoride; 1,390 p.p.m. for those drinking water with the recommended 1.0 p.p.m.; 2,570 for those drinking water with 2.6 p.p.m.; and 9,410 p.p.m. for those drinking water which contained 8.0 p.p.m. of fluoride.

The absence of any impairment of health or malformation of the skeleton or teeth of those who had consumed water containing 8.0 p.p.m. of fluoride indicates that human bone is not adversely affected when it contains levels of 7,000 to 10,000 p.p.m. These data support the wisdom of fluoridation at 1.0 to 1.2 p.p.m. in cities in the northern United States and 0.7 to 0.8 in cities in the southern states where there is a greater consumption of water.—*Nutr. Rev. (May, 1959): 133.*

Rabies Transmitted by Bat Bite

Transmission of rabies by the bite of an insectivorous bat had not been demonstrated, although the disease had been transmitted by injection of saliva taken from rabid bats.

In September, 1958, a little brown bat (*Myotis californicus californicus*) twice attacked a hunter and bit his shirt. It was captured and induced to bite 3 of 6 mice, 2 days old, which died with partial paralysis 13 and 14 days later, while the 3 unbitten litter mates remained normal. The bat was well cared for in a jar, but died seven days after capture without showing abnormalities.

The brain of 1 affected mouse was examined and contained numerous Negri bodies. Other mice died of rabies 11 to 21 days after being inoculated with brain and salivary tissues from the bat.

It has yet to be demonstrated that insectivorous bats can be true carriers of rabies without showing clinical signs of the disease.—*Science (May 29, 1959): 1490.*

Epidemiological Studies on Equine Encephalomyelitis in Maryland and Virginia

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EPIZOOTICS OF A DISEASE, probably infectious equine encephalomyelitis, had been observed in horses along the eastern coast of the United States as far back as 1831.[19] The cause of the disease was established as a virus during a 1933 epizootic[4,15] and the agent subsequently distinguished from that isolated from horses on the West Coast of the United States. Later outbreaks, involving man, horses, and birds and definitely attributable to the eastern equine encephalomyelitis (EEE) virus, occurred in the New England and Middle Atlantic states in 1938,[12-14] 1945,[1] and 1956.[10] A number of foreign countries as well as other sections of the United States have experienced epizootics as a result of EEE virus activity.[3,11,21,23,25]

On the basis of information currently available, it would appear that the virus of EEE exists primarily as an infection of wild birds and that the virus is transmitted to man and other vertebrates by arthropods which obtain blood meals from infected birds.[20] Experimental evidence indicates that infected horses rarely circulate enough blood virus to infect mosquitoes. Most investigators have concluded, therefore, that the horse is a dead-end in the chain of infection.[20]

Extensive serological surveys[2,5,16,17,22,24,27] have been conducted for St. Louis (SLE), western equine (WEE), Japanese B (JBE), and Murray Valley (MVE) encephalitis neutralizing antibodies in man, horses, and birds. Similar surveys for EEE have been less extensive. In the post-epizootic serological surveys which have been conducted for EEE neutralizing antibodies, results indicate that inapparent infections occur more frequently in horses than in man.[11,18,23,26,29]

The most severe epizootic of EEE in the Maryland-Virginia area occurred in 1933. In a personal communication, Poelma (University of Maryland) estimated that nearly 1,000 horses, mules, and ponies died at that time. Cases of EEE in these animals were observed from mid-August until first frost. During August, a hurricane struck the tidewater section of this region, resulting in flood conditions. Afterward, mosquitoes appeared in great numbers and may have accounted for the severity and perpetuation of the epizootic. Other severe epizootics have occurred in Maryland since 1933 and corresponded chronologically to those observed in the other Middle Atlantic states. Despite the greatly reduced equine population, widespread vaccination and intensive mosquito control efforts, the disease remains an important, if only sporadic, problem.

On Aug. 18, 1956, a case of suspected equine encephalomyelitis (EE) was reported from a farm near Centreville, Md. A 2-year-old gelding was observed to have a temperature of 104.2 F., paralysis of the tongue, incoordination, and oscillation of the eyeballs. A clotted blood sample was obtained. On the following day the horse died and the brain was submitted to this laboratory for examination. Since another case of EE had occurred one week previously on the same farm, all surviving animals were given two doses of commercial bivalent EE vaccine of chick-embryo origin administered one week apart. Serum specimens from a group of 9 mares were obtained four days after the initial dose of vaccine; postvaccination specimens were obtained 24 days following completion of vaccination. Blood samples were taken from a second group of younger horses prior to vaccination and one week following the final dose of vaccine.

Six other equine brain specimens from suspected cases of EE were submitted to Maryland livestock laboratories in 1956; 1 in 1957, and 1 in 1958. A serological survey of unvaccinated horses was conducted on the eastern shore of Virginia in the spring

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and early summer of 1957.* Several attempts were made in the summer of 1957 and 1958 to isolate the virus of EEE from mosquitoes.

METHODS

Virus Isolation Attempts.—Animals which had died after showing central nervous system (CNS) signs were decapitated and their heads submitted to the nearest laboratory of the Maryland Live Stock Sanitary Service. Each brain was carefully removed from the skull and representative portions placed in glycerin and refrigerated at 4 C. while others were placed in formalin or other suitable preservative for histological examination. Replicate sections were frozen at −20 C. Brain specimens were then forwarded from branch laboratories to College Park for examination.

Three-week-old Swiss mice were injected intracerebrally (i.c.) with 0.03 ml. of a 10 per cent brain suspension, previously subjected to low-speed centrifugation and, if indicated, pretreated with antibiotics.** The mice were observed for the next ten days and any that developed CNS signs during this period were killed and their brains removed and processed for storage or subsequent passage. In one isolation attempt, made during 1958, day-old chicks, as well as mice, were injected with equine brain material. Inoculum was administered subcutaneously to the chicks in 0.1-ml. amounts.

Fig. 1—Map showing locations of equine encephalitis epizootiological studies in Maryland and Virginia.

Identification of Viral Isolates.—Each viral isolate was passaged and titrated several times in mice before definitive identification was attempted. Immune serum was prepared for each strain by intraperitoneal injection of healthy, adult rabbits with a series of three doses of live virus adminis-

*The authors thank Dr. W. L. Bendix, state veterinarian of Virginia, for his cooperation in this study.

**Final concentration of 5,000 units of penicillin and 100 to 500 *gamma* of streptomycin per milliliter of suspension.

tered one week apart. The first injection represented 10 to 100 mouse lethal doses ($l.d._{50}$); the second, 10,000 to 100,000 mouse $l.d._{50}$; and the final dose, about 100 million $l.d._{50}$. One week following the final injection, the rabbits were exsanguinated and the serum was filtered and stored at −40 C.

The first virus (V-52), isolated during 1956, was used as the antigen in cross-neutralization tests with known EEE and WEE immune serum.† A known strain of EEE virus (Massachusetts strain)† was used as a control. As soon as the identity of V-52 was established as being EEE virus, its immune serum was utilized in identifying the other isolates.

Serological Tests.—All neutralization tests, including those used in virus identification procedures, were performed by combining aliquots of inactivated (56 C. 30 min.) serum with equal amounts of logarithmic dilutions of infected mouse brain material.†† Material from each aliquot was injected into groups of 5 mice—0.03 ml. i.c. into each mouse. A control titration for calculating the infectivity titer of the antigen was included and duplicate sets of mice injected.

When it was possible to obtain blood from animals showing clinical signs of EE, the serum specimens were tested for both neutralizing and hemagglutination-inhibition (HI) antibodies. Hemagglutinin was prepared according to the method described by Chanock and Sabin.[8] The HI test was conducted as previously described.[7]

Mosquito Collections and Testing.—It was not possible to collect and examine mosquitoes for the presence of EEE virus during the 1956 epizootic. In 1957 and 1958, mosquitoes were collected in areas (fig. 1) where EEE had previosuly occurred. Specimens were obtained with an aspirator, identified and sorted according to species. Pools containing up to 65 mosquitoes each were kept viable in transit by holding in ventilated boxes under layers of dampened towels. Mosquito pools were triturated in an antibiotic-containing diluent and injected into mice, day-old chicks, or embryonating hens' eggs.

RESULTS

Virus Isolations and Identification.—Of the seven equine brains examined in 1956 from clinical cases of EE, virus isolations were made from three of them. An incubation period of two to five days was observed in primary isolation attempts in inoculated mice. Cross-neutralization tests, with WEE and EEE hyperimmune serums, established the identity of all 1956 isolates as the eastern type of EE virus.

It is of interest that one equine brain which failed to yield virus showed characteristic lesions of virus encephalitis on histopathological examination. This brain was from a horse in Reisterstown, Md., a section in which EE had not been previously recognized. However, this animal had been shipped from Atlantic City, N.J., a few days before developing severe signs of encephalitis and it is likely that exposure had taken place in southern New Jersey where EE was epizootic at the time.

In 1957, the only suspected cases of EE reported to this laboratory were 2 mules, and the brain of 1 mule failed to yield the virus.

An agent was recovered from the only equine brain received in 1958. Isolation was made by inoculation of both mice and day-old chicks. The incubation period of the disease in day-old chicks was 18 to 24 hours. The agent was identified by serum neutralization and HI tests as the virus of EEE.

Testing of Acute Phase Serum.—Acute phase serum specimens, obtained from 2 horses in 1956, 1 mule in 1957, and 1 horse in 1958 had respective serum HI titers of 1:160, 1:640, < 1:10, and 1:2,560, respectively. The 2 cases in 1956 and the 1 in 1958 were confirmed cases of EE. The 1957

TABLE I—Results of Tests for Eastern Equine Encephalomyelitis (EEE) and Western Equine Encephalitis (WEE) Neutralizing Antibodies in Horses and Ponies

Location	Time of bleeding	EEE		WEE	
		No. tested	No. positive†	No. tested	No. positive
Centreville, Md.	Aug.-Sept. 1956	12	1	3	2
Centreville, Md.	April 1957	4	2	0	0
Keller, Va.	June 1957	10	1	10	0
Onancock, Va.	June 1957	18	3	18	0
Pungoteague, Va.	June 1957	20	2	20	0
Chincoteague, Va.	July 1957	17	3	17	1*
Assateague Is., Va.	July 1957	8	4	8	1**

† = >1.5 logs of neutralizing antibody; * = vaccinated in 1956 − negative for EEE; ** = also positive for EEE.

†Obtained from Major E. L. Buescher, Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, D.C.

‡Strain V-52 of the EEE virus was used as the antigen to determine the presence of neutralizing EEE antibodies. Tests for WEE neutralizing antibodies were performed using the California 26 strain of WEE virus. The latter strain was obtained from Major Buescher.

mule, whose serum was negative, was 1 of 2 mules which had died on the same Maryland farm within a week of each other. The brain from the other mule was negative on virus isolation attempts and had no characteristic lesions of EE. Since there were no confirmed cases of EE in that area in 1957, it is most probable that these cases did not represent virus encephalitis.

Additional HI tests conducted on serums from horses injected with other viruses, *viz.*, rhinopneumonitis and equine infectious anemia, have given consistently negative results.

Vaccine Study.—Tests performed on postvaccination serum specimens in two groups of horses indicated that the majority of immunized horses failed to produce measurable neutralizing antibody. In the few cases in which there was an increase in neutralizing antibody following immunization, the results are obscured by the fact that there was virus activity in the area during the time of the study.

Serological Survey.—A serological survey for neutralizing antibodies in horses and ponies showed (table 1) that 16 of 89 horses tested had significant EEE neutralizing antibodies while only 4 of 76 had antibodies to WEE virus.

Mosquito Collections and Testing.—The 1957 mosquito collections were made between September 6 and 19 and consisted of 21 pools of 65 mosquitoes per pool. They were all *Aedes sollicitans.* There were 20 *Aedes vexans,* 35 *A. sollicitans,* 1 *Aedes taeniorhynchus,* 65 *Culex salinarius,* 12 *Anopheles crucians complex,* 1 *Psorophora ciliata,* and 2 *Psorophora confinnis* collected on Oct. 1, 1958. No viruses were isolated from these mosquitoes.

DISCUSSION

The isolation of only the eastern type of encephalitis virus in Maryland is not unexpected despite the fact that the western type has been isolated from both arthropods and vertebrates in nearby New Jersey. Results of the post-epizootic survey further suggest the eastern type of virus to be more active in the Maryland-Virginia region than the western type. The few serums exhibiting WEE neutralizing antibodies were from vaccinated animals or those whose history could not be ascertained.

Failure to demonstrate a consistent neutralizing antibody response in horses following vaccination may be attributed to individual differences among animals to produce antibody, or to the limitations of the neutralization test itself. Quite possibly, horses developing only a low level of neutralizing antibody following vaccination may be resistant to natural exposure. Trials of chick embryo encephalomyelitis vaccine in horses have demonstrated a substantially lower morbidity and mortality in vaccinated horses over that of controls.[28]

It is possible that the HI test may be an advantageous supplement to the other serological test methods currently employed for confirmation of EE diagnosis. The HI test has proved useful in laboratory diagnosis of human JBE and WEE.[8,9] The HI results on the few cases referred to above are further supported by unpublished data from this laboratory (Dept. of Veterinary Science) on horses experimentally infected with EEE virus, *i.e.*, measurable HI antibodies appear prior to development of detectable neutralizing antibody. Similar results have been observed in chickens inoculated with EEE virus.[6]

Failure to isolate virus from arthropods collected in 1957 and 1958 may be attributed to low virus activity during those years and an insufficient number of collections. The year 1957 was a particularly poor time for studies of arthropod vectors because of a prevailing drought during that summer. Furthermore, the preceding year had been marked by an epizootic which would result in increased immune vertebrate hosts and thus contribute to suppression of virus activity.

SUMMARY

Two agents isolated in 1956, and one in 1958, from animals with clinical encephalomyelitis were subsequently identified as eastern equine encephalomyelitis (EEE) virus. A post-epizootic survey of horses and ponies in Maryland and Virginia revealed a significant number of animals with EEE virus neutralizing antibodies. All attempts to isolate virus from arthropods in 1957 and 1958 were unsuccessful.

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Salmonellosis in Foals and Mares

Salmonella typhimurium infection occurred in 2 studs in Denmark. Three foals died and the organism was found in the feces of 1 which recovered. Four other carriers quit excreting the organism after two months. The source of infection was probably a leaking drain which contaminated the pasture. The organism was most sensitive to chloramphenicol.—*Vet. Bull. (April, 1959): Item 966.*

Fungi in the Gallbladder of Man

Candida and Cryptococcus species were found in the bile of 21 of 33 patients with cholecystitis in Brazil.—*J.Am.M.A. (March 7, 1959): 1099.*

Surgery and Obstetrics
and Problems of Breeding

Bilateral Corneal Dermoid in a Calf—A Case Report

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ON APRIL 23, 1956, the author was called to see a newborn, purebred, Guernsey heifer calf which the owner described as having an "additional eyelid." The calf, which was extremely depressed and cyanotic, was found to have a congenital heart disease in addition. Therefore, it was killed and the head was taken for study.

After photographing the eyes, both were removed and fixed in 10 per cent buffered formalin. Pieces of the affected areas were then removed, embedded in paraffin, and sections of 8μ thickness were mounted for microscopic examination.

Fig. 1—Left eye of a 1-day-old Guernsey calf, showing extensive dermoid tumor (arrows); (A) membrana nictitans.

Fig. 2—Right eye of a 1-day-old Guernsey calf, showing enlarged lacrimal caruncle and widely distributed dermoid tumor; (A) membrana nictitans (upper lid retracted with forceps.)

The left eye contained a band of haired

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skin about 0.5 cm. wide, extending from the medial canthus to the ventral limbus of the cornea where it expanded to a roughly circular area of 1.0 cm. in diameter covering a portion of the cornea. Approximately 0.5 cm. from this area, there was another area of haired skin approximately 1.0 cm. by 1.5 cm. This skin covered approximately one third of the entire area of the cornea (fig. 1).

area of similar tissue along the dorsal medial margin of the corneal limbus about 0.5 cm. in diameter and located approximately 1.0 cm. from the larger portion of abnormal tissue (fig. 2).

The dermoid hair resembled that on the remainder of the body. The remainder of the skin, including that of the upper eyelids, was grossly normal. The lower eyelids were modified slightly at the medial can-

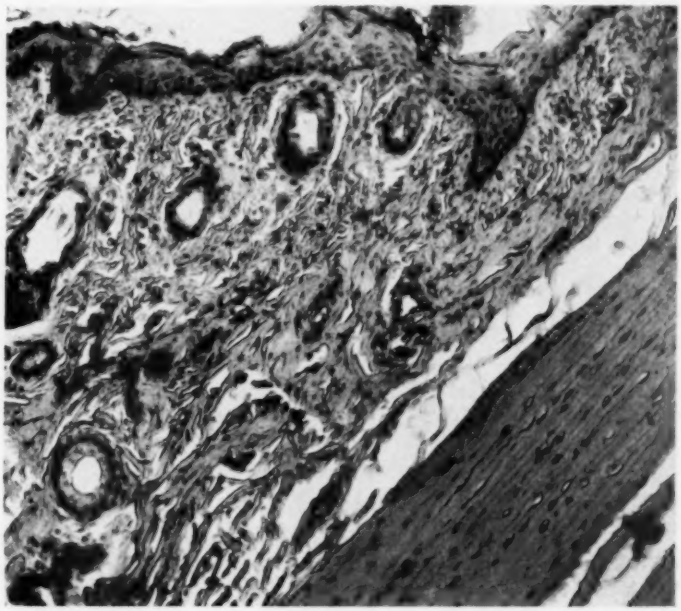

Fig. 3—Photomicrograph of edge of dermoid tumor from the left eye of a calf (see fig. 1). Notice gradation from epidermis toward normal corneal epithelium (upper right) and hair follicles and tubular glands in the tumor. The substantia propria of the cornea is at lower right. x 150.

Histological sections showed the tissue to be that of true skin containing hair follicles, sebaceous and apocrine glands, indistinguishable from surface skin in other regions of the body. The germinal layer of the stratified squamous epithelium contained pigment similar to that found in other skin areas. The epidermis blended at the margin of the lesion with the epithelium of the cornea. Aside from this area, the corneal tissue appeared normal (fig. 3).

The right eye was more severely affected. The lacrimal caruncle was greatly enlarged into a mass of haired skin about 2.0 cm. in circumference and 1.5 cm. high. From this a band of similar tissue about 1.0 cm. wide extended around the ventral margin of the corneal limbus to the lateral side of the globus oculi. In addition, there was a small

thus by the presence of the large growth of abnormal tissue, but their lateral portions appeared normal. The nictitating membranes were apparently normal. The diagnosis was dermoid tumors of the conjunctiva.

DISCUSSION

Dermoids affecting the eyes of animals have been reported for many years, including (in 1901) 24 cases in the ox, dog, sheep, pig, and cat.[6] Several authors[1,2,7] have reported that the usual site is the corneoscleral junction. One author[6] stated that dermoids are more commonly seen in the region of the lateral canthus. Some were exclusively attached to the cornea and almost completely covered this structure.[2] Usually only one eye is affected, but oc-

casionally bilateral dermoids have been reported.[4]

The embryological basis for these anomalies seems somewhat obscure. They have been ascribed to: (1) an anomaly of differentiation of the pluripotential cells lying between the optic cup margin and the surface,[5] (2) to the remains of amniotic adhesions,[8] and (3) to an invagination of ectoderm.[6] It would appear most likely that the first theory[5] is accurate. However, the theory involving an isolated island of ectoderm which is capable of developing the skin adnexae[6] is also a possible explanation.

Since the consensus is that these anomalies are frequently accompanied by other anomalous conditions, the latter should be considered carefully before initiating treatment. Attempted removal of severe dermoids, such as in this case, would be difficult, and corneal opacities would probably result. However, less severe dermoids can be treated successfully by simple surgical excision.[1,3,7]

SUMMARY

A case of severe bilateral corneal dermoids in a calf is described, with possible explanations for their formation. Bilateral cases seem to be uncommon.

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Replacement of Bovine Abomasum

Four cases of displaced abomasum in cows, 3 of them within 14 days after calving, are reported from Britain. All were treated first by casting and rocking, but only 1 responded. A second cow responded immediately after surgical replacement involving two operators.

A third cow recovered in ten days following the operation in which two men each worked with one arm through the left flank incision. The prognosis had been doubtful because the mesentery prevented the man whose arm passed over the rumen from grasping the abomasum and pulling it up on the right side when the other operator pushed it to the right side under the rumen.

In the fourth case, the two operators had the same difficulty because of the interposed mesentery, and the cow failed to improve. Later (after correspondence with Sidney Jennings of Glascow University), the incision in the left flank was reopened and the manipulation was repeated. This time, to avoid the mesentery, one operator passed his hand above the rumen toward the right hip joint until the fingers reached the abdominal wall, then moved the hand forward along the abdominal wall until he could touch the liver directly, then moved the hand downward and forward to the abomasum and beyond to the anteroventral wall of the rumen. The operator then grasped the abomasum, when it was pushed under the rumen by the other operator, and pulled it well up into the right sublumbar fossa. The cow recovered quickly and completely.—*E. P. Barrett and J. Nicol in Vet. Rec. (Dec. 20, 1958): 1206.*

Dystocia with Vaginal Rupture

A 3-year-old heifer that had been in labor several hours appeared to have a markedly swollen vulva. The calf was in posterior presentation with ventrosacral position. The cow was given an epidural anesthetic, then the calf was rotated to the dorosacral position and the hindlimbs were delivered. The left lateral wall of the vagina then ruptured posterior to the vulvovaginal junction with prolapse of 10 or 12 feet of the badly congested small intestine.

The cow was rolled so her feet were under her, her hindquarters were elevated, the calf was repelled, the prolapse reduced, the vaginal wall sutured, and the sutured area protected by manual pressure while a live calf was delivered.

The cow was given antibiotic therapy for two days and was eating normally on the third day.—*G. F. Godkin in Vet. News, Alberta V.M.A. (March, 1959): 10.*

Low Fertility in Turkeys Associated with Vibriosis

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FOR SOME YEARS, the senior author has been aware of a problem of infertility in turkeys and poor hatchability in their eggs that could not be accounted for by nutrition, genetics, environmental circumstances, flock management, or hatchery operation. Consequently, the condition is assumed to result from disease.

Any severe disease such as erysipelas may be expected to adversely affect reproductive performance. Economically, however, the most important diseases are those which only slightly disturb the general health of the individual bird or flock, but which selectively impair some phase of the reproductive cycle such as egg production, shell quality, fertility, or embryonic development. Pullorum, fowl typhoid, and the paratyphoid group of infections are well-known examples.[3] Other examples are Newcastle disease, infectious bronchitis, and chronic respiratory disease.

The Salmonella group of diseases are due to clearly defined and easily identified bacterial agents. Since all problem flocks considered in this report were classified as "pullorum-clean," and livability in the poults was excellent, this microbial group can be discounted as likely etiological agents. Newcastle disease virus can likewise be discounted, since this infection in the adult is associated with a precipitous fall in egg production,[1] which was never observed in any of the case studies. Infectious bronchitis is not known to be pathogenic for the turkey. Pleuropneumonia-like organism (PPLO) infection, or chronic respiratory disease, may be responsibie for partial reproductive failure and the organism is frequently isolated from the egg,[4,5] but the prenatal manifestations of this infection are primarily those of embryo mortality,[5] whereas the condition in question is characterized mainly by infertility, in which even initial development of the embryo does not occur. Furthermore, the incidence of PPLO infection in domestic birds, both chickens and turkeys, is great, while the present condition is restricted and localized.

Since the preceding list includes commonly known diseases which selectively impair reproduction in the turkey, the infertility condition, if infectious, must be due to some hitherto unreported agent.

The infertility problem has assumed major proportions in parts of the Pacific Northwest, but is not limited to that area. During the 1957 to 1958 hatching season, two flocks of Bronze turkeys were studied near Fayetteville, Ark. (table 1) in which fertility and hatchability were grossly subnormal. The eggs were custom hatched with eggs from flocks showing normal hatchability. The same feed was given to them as to other flocks, management practices were essentially the same, and the birds were of the same genetic origin, yet the fertility and hatchability of the eggs remained consistently low throughout the season.

TABLE 1—Fertility and Hatchability of Eggs in Two Turkey Flocks

Flock	Total eggs*	Percentage fertile**	Percentage hatched	
			All eggs	Fertile eggs
A	63,609	50.2	35.9	71.5
B	50,049	68.6	47.9	69.8

*Biweekly settings—February 20 to May 29. **Ten-day candling.

The problem in these two flocks was one primarily of infertility rather than embryo mortality. Fertility in eggs of the Bronze turkeys should exceed 75.0 per cent with 60 or more poults being hatched per 100 eggs set.[6*] The average fertility for the season in these two flocks was 50.2 and 68.5 per cent with a total hatch of 35.9 and 47.9 per cent, respectively. On the other hand, the hatch of fertile eggs was reasonably good, being 71.5 and 69.8 per cent for the two flocks.

A flock problem in infertility might reside in the male or female line. That the

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*Personal communications from turkey hatchery operators in the Pacific Northwest, Arkansas, and Virginia.

problem more likely was in the females in this case is indicated by an associated deterioration in egg shell quality. An early change is the loss of normal pigments, with the eggs becoming white like Leghorn eggs. Instances are known where flock owners have added iron salts to the ration in an endeavor to restore pigmentation. Simultaneously, the shell may lose its normal glossy sheen, become dull and roughened, and assume the texture of fine sandpaper. Other characteristics include shell wrinkling and a proportion of misshapen eggs.

Clinical manifestations, if any, in adults are obscure. There is no obvious effect upon egg production, although the matter has not been studied critically. Some owners have described a partial stupor in the birds and a "drawing up" of the wattle. However, other than the changes in shell quality and texture, manifestations characteristic of all affected adult flocks have not been described. Livability in the poults that hatch is reported to be excellent, or even greater than normal. Possibly a selection of the better-quality eggs occurs in consequence of the low fertility.

Some obscure factor of management, feeding, weather, etc. might have been responsible for the partial reproductive failure in the two flocks. However, several cases studied on the Pacific Coast by the senior author corresponded with the Arkansas condition in every important detail. The following evidences suggest that the infertility condition is of infectious origin:

1) The deterioration in egg shell quality and texture.

2) In the absence of depopulation, the condition has tended to recur annually on the same premises.

3) Breeders derived from problem-flock eggs which have been shipped a distance are reported to possess normal reproductive capacities.

4) Beyond any reasonable doubt, the continuous administration of oxytetracycline in the mash at 40 or more Gm. per ton has greatly reduced the incidence of the condition or may have overcome it completely.

Bacteriological and Other Procedures

Assuming that the condition is infectious, the etiological agent should be found within the uterus and presumably also within the egg. Since 1956, hundreds of infertile or "clear" eggs have been cultured from affected flocks using standard bacteriological procedures. The eggs have originated from flocks in the Pacific Northwest and Arkansas. A variety of microorganisms was isolated which included coliforms, staphylococci, streptococci, and Pasteurella species. None of these were regarded as being the probable etiological agent.

Having failed to isolate an agent by ordinary bacteriological procedures, other techniques were employed including anaerobic culture, culture under carbon dioxide tension, and use of semisolid agar. The two former procedures gave negative results, but from the semisolid agar cultures there was immediately isolated a gram-negative organism from a high proportion of "clear" eggs originating from the two flocks previously mentioned.

Cultural procedure has been as follows: the egg is wiped with alcohol and the air cell exploded with a hot flame. Both albumin and yolk contents are withdrawn with a pipette and deposited upon the surface of tubed semisolid medium prepared by adding 0.2 per cent agar to brain heart infusion broth (BBL). After 24 to 48 hours' incubation at 37 C., growth of the organism in question may initiate at the inoculum-agar interface, and subsequently descend into the depths of the medium.

The morphological, cultural, and biochemical characteristics of the organism are:

1) Pleomorphic; short to long, slightly curved rods, approximately 0.8 μ in diameter.

2) Motile by means of a single polar flagellum.

3) Predominately gram-negative, but somewhat gram-variable; gram-staining granules regularly present within cells.

4) Aerobic; facultative anaerobe.

5) Colonies opaque, white, crenate.

6) Broth turbid with sediment and often slight pellicle; growth regularly initiates only in consequence of heavy inoculums.

Biochemical properties of the organism are: (1) carbohydrates not fermented; (2) litmus milk unchanged; (3) gelatin not liquefied; (4) nitrates not reduced; (5) acetyl-methyl carbinol not formed; (6) indol not formed.

In consequence of the above morphological, cultural, and biochemical properties, the organism has been ascertained to probably classify in the genus Vibrio.[2]

Using the cultural method described, the Vibrio was isolated in apparent pure culture from 80 per cent of infertile eggs examined from the two flocks identified in table 1. In addition, a similar or identical organism was isolated from the infertile eggs of a flock of Beltsville White breeders located near Fayetteville. The maximum hatchability secured from any setting of eggs from this group had been 50 per cent. Finally, a Vibrio was isolated from about ten per cent of 190 hatching eggs shipped to us from a source in North Carolina. In this group of eggs, fertility measured 32 per cent on the tenth day. The flock in question was de-

scribed as possessing a maximum hatchability of 40 per cent. No further details are known.

On the other hand, Vibrio organisms were not isolated from approximately 600 infertile eggs cultured from turkeys regarded as being normal with respect to reproductive performance.

Using heavy inoculums into brain heart infusion broth, the *in vitro* sensitivity of the Vibrio was tested against a series of test drugs (table 2).

The Vibrio is seen to be highly sensitive to the tetracycline antibiotics, to erythromycin and penicillin, to dyes and to the anticoccidial, arsenosobenzene. Any one of these materials might, therefore, be regarded as a potential agent in its control.

STUDIES ON CONTROL

Over the past three years, a great number of turkey breeders in the Pacific Northwest have been medicated empirically with antibiotics, mainly oxytetracycline, in a search for control. Some of these results have been as follows.**

Operation A.—Operation A consisted of a master breeder farm of Beltsville Whites with several subsidiary multiplication flocks. Reproductive problems were first encountered in 1954 when fertility dropped near the middle of the reproductive period from about 80 to 50 per cent. The problem grew worse each succeeding year. Antibiotics were tested in a variety of ways; the best results were secured by the continuous administration of oxytetracycline in the mash. During the 1956 to 1957 season, birds in all breeding units were so medicated. Fertility as of March, 1957, for five flocks is compared with the corresponding month in 1956 (table 3).

Operation B.—Three large Broad-Breasted Bronze breeder farms were used for operation B. During the 1955 to 1956 season, nine tests during January and February showed an average fertility of 66.1 per cent. No treatment was attempted. During the succeeding season, all birds were medicated continually with oxytetracycline (50 Gm./ton of feed) beginning one month after the first egg drop. Eight fertility determinations over the same period showed an average of 81.2 per cent, 15 points higher than in the previous year.

Operation C.—Operation C employed a single unit of 2,500 Bronze breeders, and guaranteed 75 per cent fertility at transfer time. During the 1955 to 1956 season, 45 fertility determinations showed an average of 65.1 per cent. During the 1956 to 1957 season, the birds were allotted to three equal groups. Two groups were medicated continually with oxytetracycline at 50 and 100 Gm. per ton of feed, while the third group was given the drug at 100 Gm. per ton one day per week. In addition, the birds were injected at intervals with penicillin-streptomycin. Fifteen tests over the season showed an average fertility of 75.6 per cent. There were no significant differences between the three subgroups.

**Summary of field test results conducted by the senior author from 1955 to 1957, during his tenure as research veterinarian for Chas. Pfizer & Co.

TABLE 2—In Vitro Sensitivity of a Vibrio Isolated From Infertile Turkey Eggs to Test Drugs

Drug	Minimum inhibitory conc. (μg./ml.)
Chlortetracycline HCl	< 0.25
Oxytetracycline HCl	< 0.25
Erythromycin thiocyanate	< 0.25
Dihydrostreptomycin	15.6
Potassium penicillin G	0.12*
Zinc bacitracin	62.0
Furazolidone	>250.0
Arsenosobenzene	< 0.25
Brilliant green	< 0.03
Gentian violet	0.62

*Units per milliliter.

Operation D.—Operation D represents the accounts of a medium-large feed company feeding in excess of 50,000 turkey breeders. Fertility problems were formerly encountered on a spot basis in several but not all flocks. During the 1957 to 1958 season, all turkey breeder mash was fortified with 40 Gm. of oxytetracycline per ton. Reproductive performance for the season was regarded as normal according to the type and strain of bird in all instances.

TABLE 3—Comparative Fertility of Turkey Eggs from Five Flocks for March, 1956 (No Treatment) and 1957 (Treated with Oxytetracycline)

Premise	Percentage of fertility	
	1956	1957
1	45.9	85.0
2	54.2	77.2
3	50.0	75.9
4	36.9	82.5
5	51.8	70.7

DISCUSSION

The infertility condition considered in this report has been encountered and studied both in the Pacific Northwest and

in Arkansas. The Vibrio associated with the condition in the three outbreaks encountered near Fayetteville and with the North Carolina birds has not been identified in eggs from Pacific Coast flocks; however, the procedures used were not suitable for the isolation of Vibrio.

The causal relationship of the Vibrio to the infertility problem has not been proved by bird inoculation. The isolation, however, of any organism in high incidence from the interior of the egg, particularly infertile or dead-embryo eggs, is esteemed to be of pathological significance, since no microbial flora is recognized as indigenous to egg contents. In all four instances where Vibrio has been isolated from infertile eggs, the reproductive performance of the corresponding flock has been substandard.

Undoubtedly the continuous administration of oxytetracycline in the mash at concentrations as low as 40 or 50 Gm. per ton has been associated with the control, or even the disappearance of the condition. In the Willamette Valley of Oregon and adjacent areas of southern Washington, there were no known instances of abnormally low fertility in turkey breeders during the 1957 to 1958 hatching season. Essentially all flocks were medicated with antibiotics in the mash. In this same area, the malady in former years assumed the proportions of an industry disaster.

These observations permit some speculation as to the mode of drug action and the reservoir of infection. There is no known systemic infection of birds against which the broad-spectrum antibiotics will exercise any measurable degree of control when administered in as low a concentration as 40 or 50 Gm. per ton of feed; we cannot rationally assume that oxytetracycline would exert its action by being absorbed into the blood stream and transported in a therapeutically active concentration to the uterus. More probably, the etiological agent resides within the lower intestinal tract where it enters the uterus with the consequences described. Antibiotics are powerful anti-microbial agents within the intestinal tract, and against an organism of high sensitivity, as demonstrated for the Vibrio in question, the administration of even 40 Gm. per ton may be expected to eliminate the organisms from the digestive tract and thus preclude invasion and infection of the uterus.

SUMMARY

A condition of infertility in turkeys and poor hatchability of their eggs which could not be related to feeding practices, genetics, flock or hatchery management is described. Reproductive performance may be reduced 50 per cent or more. Circumstantial evidence suggests that the problem is due to a uterine infection. In four separate affected flocks, an organism classifying in the genus Vibrio has been isolated from 10 to 80 per cent of infertile eggs examined. The organism is regarded as the probable etiological agent for the poor reproductive performance.

The administration of oxytetracycline in breeder mashes at concentration of 40 or more Gm. per ton, beginning at the onset of production and continuing throughout the reproductive season, has been associated with the disappearance of the malady from affected areas.

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Stilbestrol and Avian Reproduction

New Hampshire pullets, 56 days old, were injected subcutaneously with 12-mg. pellets of diethylstilbestrol. They laid fewer eggs during the first three months of production than controls, and the eggs were significantly smaller during the first six months. However, the hatchability of the fertile eggs, and the body weight and age at which they reached 50 per cent production were not significantly affected.—*Poult. Sci. (Jan., 1959)*: *211.*

The Intraperitoneal Use of Procaine Hydrochloride with Morphine-Pentobarbital Anesthesia

THEODORE ALLEN, D.V.M.

West Hempstead, New York

A frequent serious problem associated with morphine-pentobarbital anesthesia in spaying, hysterectomy, or cesarean section is the disturbing reflex from manipulating the ovarian ligaments. This is observed as crying, abdominal pumping, and evisceration despite deep anesthesia.

This stubborn reflex interferes with surgery and is especially troublesome in small or obese animals. The condition can lead to fatal results where additional anesthesia is administered as a solution.

For the past three years, this problem has been successfully eliminated by anesthetizing the peritoneum with procaine hydrochloride. The procedure is as follows.

With the anesthetized animal in a horizontal position, an intraperitoneal injection of 2 per cent procaine hydrochloride without epinephrine (15 cc. for small dogs, 20 cc. for medium and large) is given. The bladder must be emptied—the site is midline, immediately caudal to the navel, the needle entering dorsocaudally and slightly to the right. This eliminates damage to bladder, spleen, and liver. In pyometra or pregnancy, the injection must be given with extreme caution or administered after the peritoneal cavity is exposed. The latter is the safest. After five minutes, the table can be tilted from horizontal to the desired angle for surgery.

This method is nontoxic, does not affect the general anesthesia, and never resulted in any untoward results. The reflex is eliminated most effectively and permits a smooth surgical procedure.

Dr. Allen is a practitioner in West Hempstead, N.Y.

Abortion and Stillbirths in Swine

A previous two-year survey had shown that, in Britain, the incidence of stillbirths and abortions in swine was about 4.8 per cent. Only 10 per cent of the fetuses studied yielded organisms which might be considered pathological. In another study, infection was found in 22.0 per cent. The present study involved 28 herds with abortions and 33 with stillbirths.

Cultural and serological examinations indicated that pathogens may have been involved in 33 per cent of these abortions. A few of the sows had low titers against Brucella and a few against Leptospira infections, but neither organism was isolated. In spite of special culture methods, swine erysipelas organisms were not recovered. Tests for Toxoplasma infections were negative. Other sporadic infections may have been involved occasionally.

Vitamin A deficiency may have been involved in some cases. In one herd, 4 gilts on a high-protein experimental diet failed to produce live litters, while those fed a normal ration farrowed normally. In another herd, 20 gilts abruptly changed, late in pregnancy, from succulent feed to dry, fibrous pasture, developed severe constipation and 18 delivered a number of stillborn pigs.

Other possible causes included: recent transportation in five herds; fighting between sows in two; and undiagnosed illness in one. Injury from boars was frequently mentioned as a possible cause. There was no evidence of hereditary factors.—*C. N. Saunders in Vet. Rec. (Nov. 29, 1958): 965.*

Oxytocin and the Estrous Cycle

Heifers treated with oxytocin daily for seven days after the start of estrus all were again in estrus one to four days after the end of treatment. When this treatment was started on cycle day 15, all heifers came in estrus at the normal time. When treatment was continued for 14 days, estrus occurred two to three days after the last treatment. Ovulation always followed the induced estrus.

Since the two oxytocin preparations used were free of gonadotrophic hormone, it is believed that secretion of the hormone by the anterior pituitary is regulated by oxytocin.—*Vet. Bull. (April, 1959): Item 1250.*

Oxygen for the Fetus

Research with sheep and llamas, at high altitudes where oxygen is not abundant, shows that the placenta is enlarged to provide the fetus with ample oxygen.—*Sci. News Letter (May 30, 1959): 345.*

Clinical Data

Field Trial Involving Parenteral Use of Benzathine Penicillin V on *Streptococcus agalactiae* Infections in Two Holstein-Friesian Dairy Herds

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A REPORT WAS made in 1957 on the milk and blood levels in cows, following parenteral administration of benzathine penicillin.[1] The data presented indicated the plausibility of treating udder infections via the intramuscular route. The convenience and advantages of this method are obvious. This antibiotic was found to be unstable in the milk outside of the mammary gland.[2]

Since economic and public health aspects of residual antibiotics in milk are receiving considerable attention, it was thought advisable to determine the length of time which the penicillin remains detectable in a milk sample.

An opportunity to make this study was presented when the Los Angeles County Livestock Department was considering the integration of two county institutional dairy herds; one herd free from *Streptococcus agalactiae*, the other herd not.

This study was made to determine: (1) the influence upon existing mammary infections of *Str. agalactiae* of varying doses of benzathine penicillin V* (6 million-units in a single dose; 6 million units in two equal doses given at a 24-hr. interval; 12 million units in doses of 6 million, 3 million, and 3 million units at 24-hr. intervals) in aqueous suspension, administered i.m.; (2) to compare the effect of these methods of treatment with that of udder infusion previously used on this farm (1 million units of procaine penicillin G in 50 ml. of normal saline solution per quarter each day for four days); and (3) to determine the approximate time during which the antibiotic could be detected in the milk following milking.

MATERIALS AND METHODS

The drugs were: (1) benzathine penicillin V in a single-dose syringe (5-cc. size) containing 3 million units in a stabilized aqueous suspension with 0.09 per cent methylparaben and 0.01 per cent propylparaben as preservatives, and (2) crystalline procaine penicillin G in 50 ml. of pyogenic-free normal saline solution.

The animals were Holstein-Friesian cows, 27 from a Los Angeles County institutional herd, and 9 from a commercial dairy herd in Los Angeles County. All of these cows were shedding *Str. agalactiae*.

The administration of antibiotics, the collection of milk samples, and bacteriological studies were conducted by the veterinarians of the Los Angeles County Livestock Department.

Milk samples for the detection of residual antibiotics were submitted to two laboratories.**

PROCEDURE

A composite milk sample from each of approximately 70 cows was collected in Hotis tubes and returned to the laboratory under refrigeration. The California mastitis test was conducted immediately and the milk samples placed in an incubator at 37 C. for approximately 18 hours, when the Hotis test was read and recorded.

A 2-mm. wire loopful of each milk sample was streaked onto a bovine blood agar plate containing 4 per cent blood agar base (Difco), 3 per cent defibrinated bovine blood, and 0.01 per cent aesculin. The plates were incubated for 24 hours. Streptococcus colonies which did not change the medium to green or brown were transferred to a Camp plate and incubated for 24 hours to establish their identity.

Several months previously, cultures from the institutional dairy herd had been sent to the University of California and U.S. Public Health Laboratory in Georgia

From the County of Los Angeles Livestock Department, Los Angeles, Calif.

*Manufactured by Wyeth Labs., Inc., Philadelphia, Pa.

**Dr. C. B. Ward, bacteriologist, Arden Farm Co., Los Angeles, and Tanya Gubanow, laboratory technician, Challenge Cream & Butter Association, Los Angeles.

for verification and had been confirmed as *Str. agalactiae*.

Lactation histories of, and quarter samples from, the 9 cows from the commercial herd were obtained just prior to treatment.

Composite milk samples for detection of residual antibiotics were collected in 100-ml. bottles from each of the 27 cows of the institutional herd at 12 and 24 hours after the initial treatment and were kept under refrigeration until they were delivered to the laboratories 45 miles away.

Penicillactia has been reported to be highest between 12 and 24 hours after treatment.[1] The test for the detection of residual antibiotics in milk was essentially that previously reported with 2, 3, 5-triphenyltetrazolium chloride.[3]

RESULTS AND DISCUSSION

Bacterial cultures were used to evaluate the efficacy of treatment. The results were as follows:

1) Of the 9 cows given a single intramuscular injection of 6 million units of benzathine penicillin V, *Str. agalactiae* appeared to have been eliminated in 3, but was not eliminated from 6 cows.

2) Of the 9 cows given two doses of 3 million units i.m. at 24-hour intervals, *Str. agalactiae* appeared to have been eliminated in 5, but not from 4.

3) The 9 cows given 6 million, 3 million, and 3 million units of benzathine penicillin i.m., at 24-hour intervals, had 24 infected quarters, of which the organism appeared to have been eliminated in 11. Of the 9 cows, infection was eliminated from 4—from all of their 10 infected quarters. The trial with the commercial herd followed that with the institutional herd. Quarter sampling in this herd was a better procedure because results could be more accurately evaluated. The lactation histories indicated that ten quarters in 3 cows were affected with chronic clinical mastitis; other antibiotics had also been ineffective.

4) Of the 9 cows given 1 million units per quarter daily, as an infusion for four days, the organism appeared to have been eliminated from 7, but not from 2.

Residual penicillin could be detected in the composite milk samples up to 76 hours after the initial intramuscular injection with 3 million units; 89 hours after intramuscular injection with 6 million units; and 89 hours after udder infusion with 1 million units. No tests were conducted beyond 89 hours.

Our principal interest in this field trial was to determine if *Str. agalactiae* could be eliminated from the mammary gland by the method described. Therefore, all cows shedding this organism were treated regardless of the degree of mastitis.

The 27 animals under treatment were milked as a unit separate from the remainder of the herd. The same was true of the 9 cows in the commercial dairy herd.

SUMMARY

1) By giving intramuscular injections of benzathine penicillin V, in a field trial with 27 infected cows, *Streptococcus agalactiae* were eliminated in 12 (44%); in 3 of 9 cows given a single i.m. dose of 6 million units; in 5 of 9 cows given two i.m. 3 million-unit doses at 24-hour intervals; and 4 of 9 cows (11 of 24 infected quarters) treated with 12 million units, given i.m. in doses of 6 million, 3 million, and 3 million units at 24-hour intervals.

2) This organism was eliminated in 7 of 9 cows (77%) treated by udder infusion with penicillin G_1.

Penicillin in drawn milk was detected by laboratory tests, up to 89 hours following the i.m. injections of 6 million units or mammary infusion of 1 million units.

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Tuberculosis in Goats

Of 227 goats housed with cattle, in Germany, 24 were positive to tests with bovine tuberculin. Only 2 were kept with cattle which were free of tuberculosis. Of the 8 goats slaughtered, bovine tubercle bacilli were found in 7 and avian type bacilli in 1. A bovine tubercle bacillus was cultured from the milk of only 1 of the lactating, reacting goats.—*Vet. Bull. (April, 1959): Item 938.*

Eperythrozoonosis in a Herd of Purebred Landrace Pigs

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EPERYTHROZOONOSIS is an acute febrile disease, primarily of young pigs, in which the predominating clinical signs are severe hemolytic anemia and icterus. It has been referred to as an "anaplasmosis-like disease" because of the erythrocytic inclusions associated with this condition.

In a series of studies starting in 1950, the etiological agent in this disease was revealed to be a Hemobartonella-like organism which infected the erythrocytes.[1-5] The organism was named *Eperythrozoon suis*. The disease was first reported from the hog-raising states in the Middle West, but was later reported from the South and far West. Positive identification of acute eperythrozoonosis has been made in California, Georgia, Illinois, Indiana, Iowa, Kansas, Nebraska, New York, North Dakota, Missouri, Ohio, South Dakota, and Wisconsin.[2,3]

CASE REPORT

On Oct. 13, 1958, a clinician on the staff of the ambulatory clinic was called to examine a herd of 70 purebred Landrace pigs, 10 weeks old, for a condition described by the owner as "weakness in the hindlegs." Twelve pigs in the herd showed the posterior weakness and 8 had died. These pigs were from the litters of purebred Landrace sows that were pregnant when purchased from a Georgia breeder four months previously. Shortly after weaning, the pigs were moved to an area approximately 5 miles away from the sows. The land had not been used by hogs for over seven years.

The affected pigs showed a pronounced paleness of all visible mucous membranes, a rapid heart rate, and several were observed to extend their necks as if gasping for breath. The body temperatures varied from 99.5 to 104.6 F. There was no icterus. One of the pigs with a subnormal temperature died during the physical examination. The principal lesion seen at necropsy was a severe anemia characterized by a pale, watery blood and extreme blanching of the carcass. There were multiple intermuscular and subcutaneous extravasations and massive blood clots in the stomach and intestines. The bone marrow was pale and firm.

Eight clinically anemic pigs that had elevated temperatures were brought to the veterinary hospital for further study. Hematological findings confirmed the clinical appearance of extreme anemia. The hematocrit reading ranged from 12 to 21 per cent and hemoglobin values ranged from 4.04 to 6.59 Gm. per 100 ml. of blood. Red blood cells (r.b.c.) varied from 2,500,000 to 3,600,000 per cubic millimeter. Examination of the stained blood films disclosed other features of an extreme anemia, e.g., normoblasts, pronounced polychromasia, numerous Howell-Jolly bodies, and anisocytosis. The most unusual finding was the presence of small basophilic rings and disks in or on about 5 per cent of the red blood cells. Their size and appearance closely resembled that given for *E. suis*. Only 2 of these pigs survived.

EXPERIMENTAL PROCEDURE

To confirm the diagnosis etiologically, and to compare the course of the disease in its entirety with that described in the literature under eperythrozoonosis, transmission experiments were undertaken. Two healthy Duroc pigs, 3 months old, were splenectomized prior to inoculation.

Pig. 1.—On the day following splenectomy the hemogram and temperature of pig 1 were normal. The pig was inoculated intravenously with 10 ml. of heparinized blood from a field case. A stained smear of the inoculum disclosed an infection of less than 5 per cent of the erythrocytes. On the third postinoculation day, the temperature rose to 104.4 F. but there was no significant change in the hemogram and no organisms could be found in the blood smears. Anorexia and depression were observed. There was little change in the clinical appearance or temperature of the

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pig for the next two days, but the organisms began to appear in the blood in large numbers (fig. 1).

On the sixth day, the body temperature was 105.6 F. and the proportion of infected cells had risen to approximately 90 per cent. The temperature remained above normal until the terminal stages of the disease. The packed cell volume was 19 per cent, hemoglobin value was 6.0 Gm. per 100 ml. of blood, r.b.c. were 3,140,000 per cubic millimeter, icterus index was 15, and there were nine normoblasts per 100 white blood cells (w.b.c.). Inappetence, lassitude, weakness, and anemia became progressively worse and the animal died 13 days after infection.

On the seventh day, the hemoglobin value had dropped to 2.75 Gm. per 100 ml. of blood, there were 1,860,000 r.b.c. per cmm., the icterus index was 5, and there were 35 normoblasts per 100 w.b.c. The proportion of infected red cells had declined to approximately 50 per cent. On the eighth and ninth days, the hematocrit reading did not change appreciably, but the normoblasts increased from 39 to 72 per 100 w.b.c. The number of organisms in the erythrocytes had decreased sharply and could only be found after a prolonged search.

From postinoculation days ten to 13, the hemoglobin value continued to decline and was 1.8 Gm. per 100 ml. of blood with only 2 normoblasts per 100 w.b.c. on the day the pig died. Throughout the course of the disease icterus was never clinically apparent, and with the exception of the sixth day, the icterus indices were well within the normal limits. At necropsy, the extreme anemia and some degenerative changes in the liver were the only changes that could be directly ascribed to eperythrozoonosis.*

Pig 2.—This pig, which was inoculated intravenously with 20 ml. of heparinized blood from a naturally infected field case, showed no appreciable change in temperature for the first ten days. However, the hemoglobin value dropped from 15 to 9 Gm. per 100 ml. of blood. On the eleventh day the temperature had risen to 106 F. and 95 per cent of the erythrocytes were infected. The hematological findings were as follows: hematocrit reading—19 per cent; Hb—6.10 Gm. per 100 ml. of blood; r.b.c.—3,140,000 per cubic millimeter; icterus index—3; normoblasts—14 per 100 w.b.c.

During the next three days, the body temperature dropped to 102.5 F. but the hemoglobin value had declined to 3.90 Gm.

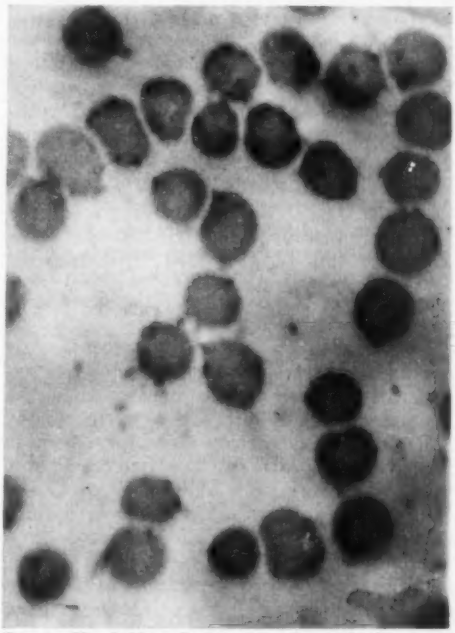

Fig. 1—Blood film of pig 1 on the sixth day postinoculation; oil immersion.

per 100 ml. blood and there were 2,180,000 r.b.c. There were 30 normoblasts per 100 w.b.c. The eperythrozoon organisms had decreased sharply and were seen only occasionally. From the fifteenth to the eighteenth day the hemoglobin value increased to 6.40 Gm. per 100 ml. of blood and the number of normoblasts had dropped to 12 per 100 w.b.c. Eperythrozoon organisms could not be found even after a prolonged search. This pig never showed icterus and the icterus indices remained within the normal range. It was killed for necropsy on the twenty-third day. Aside from anemia, no pathological changes were observed.

DISCUSSION

The nature and course of the disease encountered and reproduced left no doubt

*Blood films from pig 1 made during the acute phase of the disease were examined by Dr. H. D. Anthony of the Kansas State Veterinary College. He concurred in our evaluation of the slide and described the structures as identical with *E. suis*.

that it was the condition previously described as eperythrozoonosis by others. It was characterized in its early stages by an elevated temperature without clinical anemia, but with enormous numbers of parasites in the r.b.c. and plasma.

Both experimentally infected pigs survived the acute phase of the disease and entered the anemic phase, which was accompanied by a sharp drop in the temperature and a marked reduction in the number of parasites present in the circulating blood. Subsequently, during a regenerative phase, great numbers of immature erythrocytes appeared in the blood. This stage may continue for an extended period, as in pig 2. This pig, which had passed the acute phase by postinoculation day 13, showed regenerative r.b.c. changes and a slowly but steadily progressing anemia until killed on day 23. These stages are in close agreement with accounts of the disease in the literature.[2-4,6]

Apparently the naturally infected animals when first seen were entering the regenerative phase of the disease when the anemia was at its height and the organisms had almost disappeared from the blood. It appears that death can occur at any point during the infection. In this respect, our observations are in agreement with published accounts of the disease, as in the naturally infected animals and the experimental pigs, deaths occurred in the regenerative phase of the disease. Icterus was not present in any of the naturally or the experimentally infected pigs; however, it has not been a constant feature of the disease.[1,4,5]

SUMMARY

A naturally occurring case of eperythrozoonosis in a herd of purebred Landrace pigs is described. An account is given of the course of infection in 2 splenectomized pigs inoculated with fresh blood obtained from field cases during the latent phase of the disease. The natural and the experimental disease is discussed with special reference to comparison with accounts of the disease in other areas.

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A Virus Enteritis in Italian Swine

In 1956, a new form of infectious enteritis was observed in swine of the Po Valley. It commonly affected feeder pigs. It spread rapidly, and affected 18 to 82 per cent of the animals in the herds. The illness, which lasted three to ten days, was characterized by a profuse diarrhea which was not hemorrhagic or foamy. Mortality was low.

The disease was readily transmitted with filtered feces; the incubation period was 24 to 72 hours. It could not be transmitted to small laboratory animals or embryonating eggs. Survivors were immune and their serum contained neutralizing antibodies. Clinically, in the early stages it could be confused with hog cholera but it more closely resembled transmissible gastroenteritis.—*G. Gualandi in Die Vet.-med. (Jan., 1959): 60.*

Prepartum Iron and Pig Anemia

Sows injected intramuscularly with 10 ml. of iron-dextran solution, two weeks before farrowing, produced pigs which maintained higher hemoglobin levels than did pigs from untreated dams or from dams treated four weeks prior to farrowing. The latter injections had little effect.—*J. Anim. Sci. (Feb., 1959): 415.*

Tetanus as a General Infection

It was proved experimentally in laboratory animals that infection with tetanus spores is not always limited to the point of inoculation. Within 30 minutes of injection, spores were found in internal organs. Tetanus organisms can also penetrate from the intestinal tract. Tetanus antitoxin may be less effective in various mixed infections, so therapy against the other organisms must also be considered.—*J. Kalich in Die Vet.-med. (Jan., 1959: 48.*

Sodium Pentachlorophenate as an Ovicide for Controlling Ascariasis in Swine

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Columbia, Missouri

IN RECENT YEARS there has been considerable progress in efforts to control *Ascaris suum*, the common roundworm of swine. The piperazines have proved to be effective and safe ascarifuges.[3,6] More recently, hygromycin B has shown promise in destroying the fecundity of the female worm. Assuming that this product could be fed to all swine on a farm for several successive years, it might eliminate the ascarid as a major parasite threat.[4,13] However, neither product affects the ascarid until it has made its sojourn in the liver and lungs and produced much of the ultimate damage.

The swine ascarid egg is known to be highly resistant to natural adverse environmental factors. They have been found to remain viable in the soil from one to five years.[2,8,14] For this reason it appears that soil sterilization might play a vital role in controlling the swine ascarid.

Several investigators have demonstrated that chemicals can be applied as effective soil larvicides. Sodium pentaborate tetrahydrate was used to control the swine kidney worm[1] and sodium borate to destroy the larvae of the canine hookworm.[5] However, the parasites that reach their infective stage inside the heavy protective wall of their shells are not susceptible to the commonly used larvicides.

Until recently such parasites as the ascarids, whipworms, cecal worms, and pinworms could not be effectively controlled by chemical ovicides. After it was found that sodium pentachlorophenate (Santobrite*) was ovicidal to ascarid eggs, in concentrations as low as 10 p.p.m., it was used in a preliminary field test which indicated that sodium pentachlorophenate sprayed on the litter gave about 65 per cent control of the poultry ascarid.[11]

The purpose of this paper is to present the results of tests designed to evaluate sodium pentachlorophenate as an ovicidal agent for swine ascarid eggs in the soil.

MATERIALS AND METHODS

Three tests, somewhat similar in design, were conducted. The first two tests used sows and baby pigs; the third test used pigs, 6 weeks old.

TABLE 1—Plot Arrangement and Time of Tests 1 and 2, Using an Ovicide to Control Ascariasis in Swine

Plot No.	Soil treatment	Test 1		Test 2	
		Animals	Time on test	Animals	Time on test
A	Sodium pentachlorophenate, 12 oz. in 2 gal. water, per 100 sq. ft. soil	1 sow 10 pigs	4-15-57 to 7-1-57	1 sow 9 pigs	10-17-57 to 11-25-57
B	2 gal. water only	1 sow 8 pigs	4-15-57 to 7-1-57	1 sow 8 pigs	10-17-57 to 11-25-57
C	Sodium pentachlorophenate, 12 oz. in 2 gal. water, per 100 sq. ft. soil	1 sow 5 pigs	4-15-57 to 7-2-57	1 sow 8 pigs	10-17-57 to 11-26-57
D	2 gal. water only	1 sow 7 pigs	4-15-57 to 7-2-57	1 sow 9 pigs	10-17-57 to 11-26-57

Four test pens (each 256 sq. ft. in area), in a lot used for hogs for 30 years, were arranged in a contiguous row with gradual drainage toward the front. Heavy sheet metal panels were extended 8 inches into the soil at the lateral partitions of each pen so that cross drainage could not occur. A shed-type hog house, an automatic feeder, and a waterer were placed in each pen. A commercial sow and pig meal was fed throughout all tests.

Crossbred sows were used for all tests and were bred to farrow as near the same date as possible. Numerous fecal examinations, made during pregnancy, indicated that none of the sows were carriers of *A. suum*.

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*Santobrite is produced by Monsanto Chemical Co., St. Louis, Mo.

TABLE 2—Effect of a Soil Ovicide on the Number of Liver Scars Due to Migrating Ascarid Larvae (Test 2)

Lot No.	Soil treatment	No. pigs in pens	Total scars*			Standard deviation
			Minimum	Maximum	Average	
1	Treated	9	10	33	17.6	8.0
2	Untreated	8	35	101	67.5	21.0
3	Treated	8	17	69	37.3	17.56
4	Untreated	9	54	77	67.3	8.81

*A count was made of the liver scars within the area of a circle having a 2-inch diameter on each of the four major liver lobes; these four counts were totaled to give the liver scar numbers as shown above.

Approximately three days before the farrowing date, the sows were scrubbed vigorously with hot water and a detergent to remove as many ascarid eggs as possible from their skin. They were placed in concrete-floored farrowing pens which had been thoroughly scrubbed, using both live steam and hot water with detergents.

Two to three days after farrowing, the sows and pigs were hauled to the previously prepared test pens.

Sodium pentachlorophenate solution was applied to the soil in two pens seven to ten days before the pigs were placed on test. Approximately one half the solution was sprayed on the surface of the soil which was then sprinkled with water and allowed to soak for one hour or longer. The soil was then pulverized with hand tools to a depth of 2 inches and the remainder of the solution was applied. The soil was again sprinkled to moisten at least the first 3 or 4 inches of the top soil.

Test 1, Spring, 1957.—In the first test, 30 pigs were used on the natural, undisturbed soil of the old hog lot (table 1). The sows were removed from the pens at the end of six weeks. On July 1

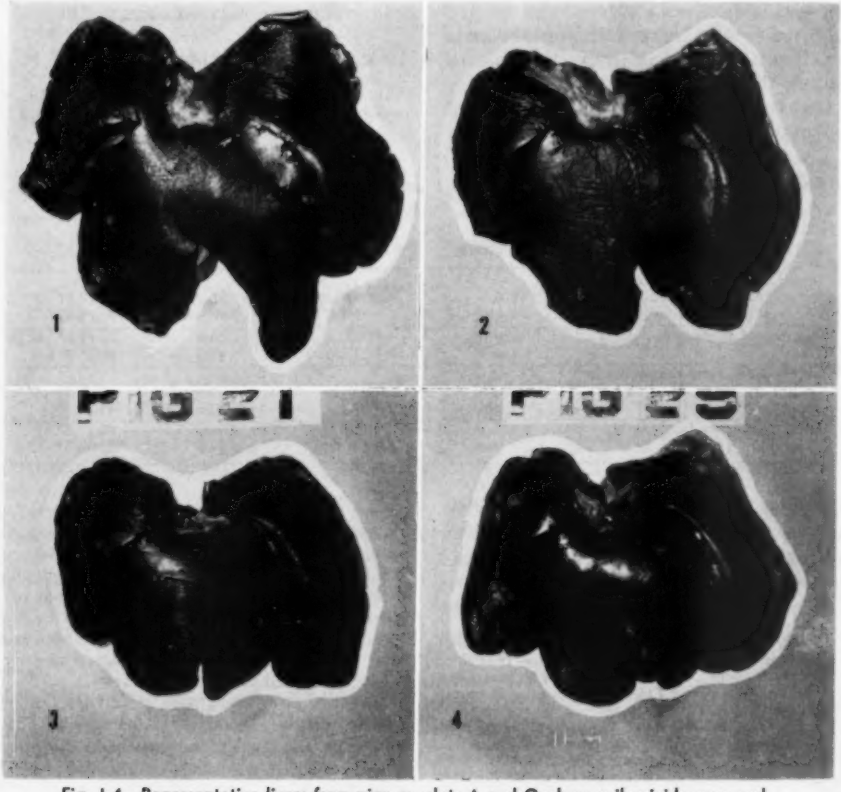

Fig. 1-4—Representative livers from pigs on plots A and C where soil ovicide was used.

and 2, when the pigs were 11 weeks old, all were killed and examined for parasites and for pathological changes indicative of ascarid invasion.

Test 2, Fall, 1957.—Soon after the first test was completed, the experimental plots were reinfested with ascarid eggs by placing 2 pigs with moderate to heavy ascarid infections in each pen. The 8 pigs were then rotated at the end of each week so that each group of 2 pigs was on each plot for a total of 14 days. These pigs were removed late in August and the pens were vacant until mid-October when the second test was initiated.

The procedure of the second test (table 2) differed from the first test only in that the pigs were killed for necropsy when 5 weeks old.

Test 3, Summer, 1958.—This test was designed to study possible economic benefits from treating ascarid egg-infested soil with sodium pentachlorophenate. Six-week-old pigs which had been raised as nearly ascarid-free as possible were placed on the test plots and kept there several weeks in order to compare the benefits of soil treatment versus nontreatment. The rate of weight gain and the feed conversion ratio were used as the major criteria for evaluation.

The same plots, means of recontaminating the plots, and methods of soil treatment that were described for test 2 were used in test 3. One major difference, however, concerned the moisture content of the soil. During July of 1958, almost 16 inches of rain fell on the test plots. At the time the sodium pentachlorophenate was applied, the soil and manure in the test plots appeared to be in a state of complete water saturation.

RESULTS AND DISCUSSION

Test 1.—When these pigs were necropsied, after 11 weeks on the soil plots, only a few ascarids were found in their intestinal tracts, and there were as many in pigs on the treated as on the untreated plots. However, the pigs on the treated plots had approximately one half as many liver scars as those on the untreated plots. There appeared to be no correlation between the degree of liver scarring and the number of parasites present.

Weight gains for pigs on the treated

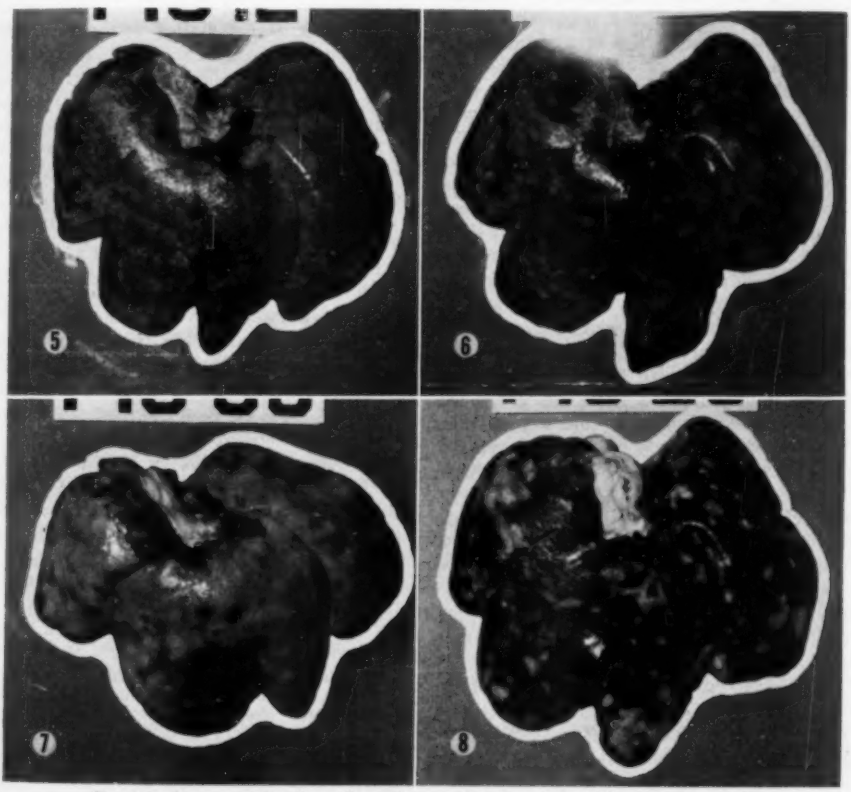

Fig. 5-8—Representative livers from pigs on plots B and D which were untreated.

plots were considerably higher than for the control pigs. The 15 pigs on the treated plots gained a total of 652 lb., while the 15 on the untreated plots gained 522 lb.

Because of the greatly reduced number of liver scars found in the pigs on the treated plots, it appeared that the ovicide had markedly reduced the number of viable eggs available to susceptible pigs. According to one report,[10] the number of liver scars found at necropsy can be used as a crude measure of the degree of ascarid infection the animal has recently acquired. Possibly the dearth of ascarids in the intestines could be attributed to an acquired immunity developed by the pigs. Also, parasites of the intestines are likely to be flushed out as long as the host is on a milk diet.[7,12] While these pigs had been weaned for about five weeks, it is possible that the milk helped prevent the establishment of early infections.

Test 2.—To verify the results of the first test with regard to the liver lesions and to see if more of the immature ascarids could be found in the intestinal tracts of the pigs at 5 weeks of age, this second experiment was conducted. It seemed that the presence or absence of immature ascarids would serve as a better criterion for evaluation of soil treatment efficiency than the liver-scarring effect.

Pigs from the control plots had approximately three times the number of liver scars as those from the treated plots (table 2, fig. 1-8). Many of the livers from the control pigs were greatly swollen, were almost a solid mass of scars, and were covered with a fibrinous film. These results were in agreement with the findings of the first test and indicated that the gross ascarid egg infestation of an old hog lot can be reduced by as much as 65 per cent by one application of sodium pentachlorophenate.

However, only 15 immature ascarids were found in all 34 pigs of test 2. In view of the severe liver scarring, especially in the pigs from the untreated plots, it appears that the milk diet, or an early acquired immunity, or both, prevented the intestinal stages from developing.

Test 3.—No difference was noticed in the rate of gain, at the end of two- and four-week periods, between the pigs on the treated and untreated soil. The feed conversion rates were essentially the same. At the end of four weeks, 2 pigs were selected for necropsy from each of the four pens. Moderate liver scarring was evident on the livers of all 8 pigs. There was no discernible difference in numbers of liver scars between pigs from treated and untreated plots. The test was continued for four more weeks with no indication that any appreciable control of ascariasis had been obtained.

The reasons for poor results in the 1958 tests are not known. Possibly the extreme water saturation of the soil at the time of ovicide application so diluted the chemical that it did not reach the ascarid eggs in sufficient concentration to be lethal. Mud and sunlight quickly reduce the concentration of sodium pentachlorophenate in water when it is used as a molluscacide.[2] Obviously, further investigations are needed to determine the effect of various soil conditions on the efficacy of sodium pentachlorophenate as a soil ovicide.

SUMMARY

The ovicidal efficiency of sodium pentachlorophenate against swine ascarids was studied by controlled critical experiments utilizing susceptible young pigs on plots heavily contaminated with ascarid eggs.

In two tests a single application of the ovicide (12 oz. per 100 sq. ft.) was applied to the soil. Liver scars were reduced between 50 and 70 per cent in all pigs on the treated plots.

Few ascarids were found in the intestines of any of the pigs. There was no correlation between liver scarring and the few immature ascarids that were present. It is postulated that the absence of ascarids in the intestines can be attributed to the milk diet or to an immunity acquired early, or to both.

A third test conducted in the summer of 1958 in an attempt to demonstrate that increased gains and feed efficiency would result from use of an ascarid ovicide failed to duplicate the results of the first two tests. A possible explanation for this discrepancy is discussed.

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The Survival of Foot-and-Mouth Disease Virus in Cured and Uncured Meat

Tests were conducted to study the survival of foot-and-mouth disease virus in cured and uncured meat. Meat samples were tested in 37 experimental groups of cattle or a total of 72 test animals. Comparable and anatomically identified samples were taken from the carcasses of infected donor cattle which had been slaughtered 33 to 35 hours after inoculation with foot-and-mouth disease virus, type A, strain 119, at a time when viremia was established and secondary lesions were beginning.

The meat samples were tested fresh, after ripening (stored 72 hr. at 4 C.), and after storage in wooden barrels at 1 C. for 16, 30, and 50 days. Each of the latter stored meat samples included barrels of cured meat (ripened and salted) and barrels of unripened, unsalted meat. Samples of muscles, blood, and lymph nodes were tested in each instance. Infective virus was demonstrated in all fresh samples; in ripened blood and lymph node samples; in unripened, unsalted lymph node samples stored 16, 30, and 50 days; and in cured lymph node samples held for 30 and 50 days.

Carcasses tested after storage at 4 C. contained demonstrable virus in rib bone marrow at 14, 60, and 73 days, and in lymph nodes, blood, and muscle samples at 60 days. Muscle samples taken after 73 days of storage were negative.

The chemical changes that occur during the ripening of meat inactivate the virus in muscle tissue but, apparently, they do not appreciably affect the virus in lymph nodes, large blood clots, or bone marrow. In transverse sections of muscle tissue taken from rapidly chilled carcasses, the pH readings show a general increase in acidity from the superficial toward the deep areas. For nearly 48 hours, conditions are more favorable for virus survival in the superficial portions of the carcass.

It was concluded that meat from animals infected with foot-and-mouth disease was not rendered free of the virus by the usual commercial procedures of ripening, boning, salting, and storage. The study showed that the virus could survive in certain tissues customarily included in such meat.—*G. E. Cottral et al.: The Survival of Foot-and-Mouth Disease Virus in Cured and Uncured Meat. Am. J. Vet. Res., 20, (Nov., 1959: In press.*]

Zinc Toxicity for Weanling Pigs

Ninety-two pigs, at the University of Illinois, were fed zinc carbonate for 42 days, at levels varying from 0 to 0.8 per cent of the corn-soybean oil meal ration. Addition of 0.1 per cent of zinc supplement was the maximum tolerated. Higher levels produced signs of zinc toxicosis characterized by depressed appetite and rate of gain, arthritis, gastritis, catarrhal enteritis, congestion of the mesentery, and by hemorrhages in the ventricles of the brain, lymph nodes, and spleen. On high levels of zinc, death often occurred in 21 days. Hemoglobin levels were unaffected. Calcium added at 1.0 per cent of the ration had no effect on zinc tolerance or toxicosis.—*J Anim. Sci. (May, 1959): 836.*

Editorial

Congenital Dysplasia of the Hip in Dogs

Guest Editorial

The results of research generally require some time to filter through to the practitioner even though *Time* and the *Reader's Digest* often inform (sic) the patient before the doctor has time to study his journals. There is little doubt, however, that congenital hip dysplasia (HD) of the dog is a widespread, often crippling, inherited disease. In dealing with it, confusion, misunderstanding, and mistrust of veterinarians have developed, due mostly to intense interest in HD among dog fanciers.

Good Radiographs Essential

Pelvic radiography is not difficult, but it does take patience, knowledge, and equipment, especially since dogs commonly involved are often of the larger breeds. Many practitioners have been called upon to do radiography for which they were not equipped and to interpret radiographs in a subject requiring specialized knowledge and experience which they have not acquired. Informed dog fanciers, either loath to believe the truth about their favorite breed or unwilling to believe an untruth, have often demanded radiographs for interpretation by "experts." Such consultation would be more judicious and proper if radiographs *properly marked* with date and film number would be referred by the veterinarian to a veterinary colleague, who then could render a written opinion, in duplicate, for the fancier if desired.

Since one good radiograph is probably all that one will get of a sore-hipped, large breed dog, it should be taken in the most desirable position. The subject should be placed on its back, the hindlegs pulled completely backward (in the position of a man standing), the mid back being supported by sandbags or held up off the x-ray table by an attendant so that the long axis of the pelvis is parallel to the film. The trochanters and femoral necks are then projected onto the film in profile, rather than foreshortened or superimposed upon each other.

Misunderstandings Occur

Many veterinarians must shoulder personal blame for lay criticism, since many entirely unreadable radiographs have been mailed for "expert" opinion. If the radiograph is then pronounced to be not of diagnostic quality, the dog, the fancier, the practitioner, and the "expert" all have suffered. It would certainly be as bad for the consultant to offer an opinion when the film was unreadable as it would be for the practitioner to offer an opinion not based on factual knowledge, for the fancier might take the "evidence" and ask for corroboration from someone who did have the correct answers. "Shopping" for opinions with a radiograph goes beyond the community—it can become national or even international. Interested fanciers may even go hundreds of miles with the dog itself if they are suspicious of the opinion rendered.

As a Matter of Record

We disagree with two contentions made in articles on HD in this issue of the Journal. Snavely's observations, as well as those of Hart (quoted by both authors), mitigate Henricson and Olsson's contentions that this disease should be called "acetabular" rather than "hip" dysplasia. All structures of the hip are involved in its primary development, as well as all showing secondary changes. Better reasons should be presented before again altering the name of this disease, already "luxuriantly" supplied with terms.

Second, lest the contention that HD occurs more frequently in females than in males become too fully accepted without greater evidence, it is worth noticing that of 122 dogs with this disease examined at the Angell Memorial Animal Hospital in the last full year (1958), 64 were males and 58 females. At this writing, more than 700 diagnoses of congenital hip dysplasia have been made at this hospital, and only exceptionally have whole kennels of dogs been radiographed.

Troublesome to the writer are the shades of "normalcy" and deviations among good, sound pelvises that might not be considered "perfect." Must every man be a physical Johnny Weismuller to be normal? Here lies the reason for our use of the word "taint." In breeds commonly afflicted with HD, there are many borderline cases. What of the quite spherical but shallow acetabulum

with a spherical and good fitting femoral head? Such hips often show considerable spur development in middle age or beyond. What about the dog that does not quite meet the criteria we set for the diagnosis of HD? And what effect does this question have on inheritance studies? Both the Henricson and Snavely reports must be evaluated in this light. Veterinarians must be aware of the variations in normal appearance between the "giant" and the "normal" breeds, or among such breeds as Pug, Pekingese, and English Bulldog. They must also be acquainted with the normal appearance of the immature pelvis, which to the uninitiated can look quite "imperfect." Also, it should be most emphatically understood that this disease is not restricted to the German Shepherd breed. At least 40 breeds are affected, and quite likely Newfoundlands, Great Pyrenees, and English Bulldogs have it in greater statistical frequency than the Shepherd.

Finally, in consideration of the complexities of the disease and of the many breeds involved, even of other species, e.g., cattle and rabbits, this is an important veterinary problem; let us not muff it, nor hand it to the laymen, nor to other professions. —*Gerry B. Schnelle, V.M.D., Angell Memorial Animal Hospital, Boston, Mass.*

Dextran as a Plasma Expander

The term "dextran" was first used to describe the gums of carbohydrate nature which were formed in the juice of sugar beets and grapes. Later, Pasteur demonstrated that they were the result of microbial action. As produced for use as a plasma expander, dextran is a polysaccharide resulting from enzymatic action of a Streptococcus on sucrose to form a high molecular weight polymer of glucose. When first used, there was a high incidence of allergic reactions, but this seldom occurs now.

When used intravenously, dextran does increase bleeding time, but not to a degree of surgical significance. The larger molecules circulate in the blood stream for up to 24 hours before they are broken down and excreted or metabolized. Blood volume gradually subsides, with 50 per cent of the dextran remaining in the circulation after six hours. It is a valuable resuscitative agent for immediate use while blood or blood derivatives are being obtained. Its place is in the emergency room, not the storeroom.—*Am. J. Surg. (May, 1959): 593.*

Whole Blood Transfusion Rate

In answer to a query, it is stated that the maximum rate of transfusion of whole blood in man is considered about 250 ml. per minute, the amount that can be forced through a 15-gauge needle. Several difficulties that can result from rapid administration are: citrate intoxication (rare); thrombocytopenia with serious clotting; and overloading the circulation resulting in pulmonary edema.—*J.Am.M.A. (April 4, 1959): 1690.*

Tetanus Causes Spinal Fracture

A case of tetanus in a 16-year-old boy was complicated by compression fractures of the thoracic vertebrae due to muscular spasms. This is a frequent complication in childhood tetanus. The sustained muscular contraction plus the tetanic spasms combines to produce fractures of these spongy, relatively delicate cubes of bone. The apex of the thoracic kyphosis acts as the fulcrum. The patient recovered.—*J.Am.M.A. (May 23, 1959): 455.*

Current Literature

Abstracts

Chicken Embryo Response to Avian Pathogens

The viruses of avian encephalomyelitis (AE), synovitis, Newcastle disease (ND), infectious bronchitis (IB), and avian pleuropneumonia-like organisms (PPLO), and the agent of avian infectious hepatitis (AIH) were inoculated into chicken embryos for the purpose of determining the pathological response of this host system.

Killed and exsanguinated embryos were examined at 24-hour intervals for 11 days postinoculation and subjected to histopathological examination at eight cross sectional levels.

Avian encephalomyelitis caused atrophy of the embryos and, microscopically, neuronal degeneration in the ventral horns of the spinal cord and dorsal ganglia. Skeletal muscles showed extensive muscular dystrophy and the CNS edema, encephalomalacia, and terminal internal hydrocephalus.

Synovitis produced splenomegaly and hepatomegaly, marked perivascular reticular cell proliferation in the liver and, occasionally, in the gizzard and skeletal muscles.

Pleuropneumonia-like organisms, hemagglutinating and nonhemagglutinating strains, produced widespread heterophilic perivasculitis, most prominent in the liver. Livers of AIH-infected embryos showed a greenish cast and, microscopically, focal necrosis, regenerating liver cord cells about the hepatic vessels, occasional aggregates of heterophils accompanied by bile duct proliferation, and bile stasis. The ND and IB viruses did not produce consistent microscopic lesions in the embryo. The pathological response of the chicken embryo is considered contributory to the differentiation of certain avian pathogens.—[*D. Roy Casorso and Erwin L. Jungherr: The Response of the Developing Chicken Embryo to Certain Avian Pathogens. Am. J. Vet. Res., 20, (May, 1959): 547-557.*]

Vaccination of Sheep with X-Irradiated Larvae of Haemonchus

This paper describes the application of the principle of partially inactivating infective helminth larvae by x-irradiation, already successfully employed to produce a vaccine against *Dictyocaulus viviparus* infections, to the trichostrongyle stomach worm, *Haemonchus contortus*.

Batches of *H. contortus* larvae were treated with 10,000, 20,000, 40,000, 60,000, and 100,000 roentgens, respectively. These larvae were then given orally to sheep.

Two animals from each group were killed 37 days after infection to assess the degree of inactivation obtained. Treatment with 10,000 and 20,000 did not produce significant reductions in worm burdens although the other levels did. The remaining 5 sheep in each group were later challenged with 8,000 normal larvae and all were killed after 45 days. The control group had a mean burden of 2,042 worms, the 40,000-r group 200 worms, and the 60,000-r group no worms.

It is considered that the optimal range for vaccine production is 40,000 to 60,000 r. The 100,000-r group, on challenge, had a mean worm burden of 442 worms and it is probable that this radiation level produces too high a degree of inactivation to allow the larvae to penetrate the stomach wall and elicit a suitable degree of antigenic effect.

The applications of this principle to field vaccination and to the study of the mechanisms of helminth immunity are discussed briefly.—[*W. F. H. Jarrett, F. W. Jennings, W. I. M. McEntyre, W. Mulligan, and N. C. C. Sharp: Studies on Immunity to Haemonchus Contortus Infection—Vaccination of Sheep Using a Single Dose of X-Irradiated Larvae. Am. J. Vet. Res., 20, (May, 1959): 527-531.*]

Newcastle Disease in Chickens

Newcastle disease virus (NDV) was not isolated from the aqueous humor of 127 birds vaccinated with live or killed vaccines, or both, when 2 to 6 weeks old and challenged intratracheally when 18, 20, or 22 weeks old. However, HI antibodies were demonstrated in the aqueous humor of each of the 34 birds tested.

The virus of ND was isolated from the aqueous humors of the eyes as well as from the tracheal fluid of naturally infected birds that were showing respiratory and nervous signs. It was not isolated from birds that were showing nervous signs only.—[*A. H. Dardiri, V. J. Yates, P. W. Chang, and D. E. Fry: Newcastle Disease Virus in the Aqueous Humor of Chickens. Am. J. Vet. Res., 20, (May, 1959): 545-546.*]

Foot-and-Mouth Disease Antibody

Swedish cattle, previously unexposed to foot-and-mouth disease virus (FMDV), were inoculated subcutaneously with 15 ml. of type A_5 or type O_1 FMD vaccines prepared by the Schmidt-Waldmann method. Maximal complement (C')-fixing antibody titers were obtained in two to three weeks and the antibody was detectable from 14 to 56 days after primary vaccination. Revaccination in 23 days resulted in increased antibody production which exceeded that following primary vaccination. Serums from 2 of 36 normal cattle tested showed low levels of C'-fixing activity with the type A_5 antigen. Intradermolingual challenge with active virus in vaccinated animals whose serums no longer had C'-fixing activity resulted in generalized infection. Vaccinated cattle which were challenged with active virus at a time when antibody was still demonstrable in their serums were refractory to infection. On the basis of these findings a relationship between presence of C'-fixing antibody and resistance to FMD infection has been postulated.—[*Americo Alvin Marucci: Detection of Foot-and-Mouth Disease Antibody in Vaccinated Cattle by Direct Complement Fixation. Am. J. Vet. Res., 19, (Oct., 1958): 979-984.*]

THE NEWS

Dr. Leslie M. Hutchings

1915 – 1959

★Leslie M. Hutchings (MSU '40), 43, former head of the Department of Veterinary Science at Purdue University and first dean of its newly-established School of Veterinary Science and Medicine, outstanding veterinary pathologist, and internationally known authority on brucellosis, transmissible gastroenteritis, and other swine enteric diseases, died on July 22, 1959, at Billings Hospital, Chicago, following a prolonged illness.

Born at Portland, Maine, Sept. 13, 1915, Dr. Hutchings received his preliminary education in the local schools and then entered the University of Maine, at Orono, from which he received his B.S. degree in 1937. He was awarded the D.V.M. degree from Michigan State College (now University) in 1940 and an M.S. degree in 1942. He was an assistant in pathology from 1937 to 1940 and then served as research veterinarian until 1942 while studying for his M.S. degree.

In 1942, Dr. Hutchings accepted appointment as associate pathologist in the Department of Veterinary Science at Purdue. He was awarded a Ph.D. degree in pathology and promoted to professor in 1947. He became head of the Department in 1950, succeeding the late Dr. C. H. Donham. In 1947, he won the Sigma Xi Research Award and also was named "Outstanding Young Man of Indiana" by that state's Junior Chamber of Commerce.

He was director of the Indiana State Animal Disease Diagnostic Laboratory for a number of years and was a member of the Indiana State Livestock Sanitary Board. He was a member of the National Research Council's committees on animal health, brucellosis, and leptospirosis; a member of the panel of experts on brucellosis of the World Health Organization, FAO, since 1950; also chairman of the U.S. Livestock Sanitary Association Committee on Transmissible Diseases of Swine in 1950, and of the National Committee for Eradication of Hog Cholera from 1950 to 1953.

Dr. Hutchings joined the AVMA in 1940, was elected to its Executive Board from District III (Indiana, Illinois, Wisconsin) in 1953 for a five-year term, and served as chairman of the Board in 1957-1958. Prior to that, he had been secretary of the Section on Research in 1950-1951 and its chairman in 1951-1952. He had also served the Association notably in other ways, especially in

Dr. Leslie M. Hutchings

Congressional hearings on matters relating to the veterinary profession and animal disease control.

He was also a member of the American Society of Bacteriologists, the Conference of Research Workers in Animal Diseases of North America, the International Congress on Tropical Diseases and Medicine, the Indiana Veterinary Medical Association, and other professional and scientific bodies, including the honorary scientific Society of Sigma Xi.

Surviving are his widow, Mrs. Mary Bruce Hutchings; three sons, 12, 15, and 17 years old; and his parents.

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Group of U.S. veterinarians and their wives at Idlewild International Airport, N.Y., on May 17, prior to take-off for the XVIth International Veterinary Congress in Madrid.
Left to right—Mr. E. L. Leenhouts, Travel Service Bureau (which arranged Pre- and Post-Congress travel and tours for the U.S. Committee-AVMA); Dr. Salo Jonas; Dr. and Mrs. E. A. Weidenbener; Mrs. and Dr. A. J. Manzoni; Mrs. and Dr. H. S. Dulman; Mrs. A. R. MacDonald; Mrs. and Dr. W. H. Riser; Dr. MacDonald; Miss Rosalyn Zirlin; Dr. Kenneth McEntee; Dr. V. H. Miller; Mrs. C. M. Christy; Dr. A. H.

Further Report on XVIth International Veterinary Congress . . .

The convening of the XVIth International Veterinary Congress, henceforth to be known as the World Veterinary Congress, in Madrid, was notable in many respects.

Madrid was a fine setting, the facilities were good, the Organizing Committee did an excellent job in arranging and carrying out the multitude of details essential to a successful and enjoyable congress, the weather was pleasant, and the hospitality ample and gracious.

The Opening Session

The formal opening of the Congress on May 21 (*see* the JOURNAL, July 15, 1959, pp. 132-133) featured brief addresses by eight speakers: Prof. Dr. Pedro Carda, secretary-general, and the Hon. Sr. E. Alcazar, president, on behalf of the Organizing Committee; Prof. Dr. Charles Bressou, France, for the French-speaking countries; Dr. W. A. Hagan, chairman of the U.S. Committee, for the English-speaking countries; Prof. Dr. Kurt Wagener, Germany, for the German language countries; Prof. Dr. Jac. Jensen, Netherlands, secretary-general of the I.V.C. Permanent Committee, and Prof. Dr. W. I. Beveridge, Australia, its president; the Hon. Sr. A. Campano, director-general de ganaderia, and the Hon. Sr. C. Canovas, minister of agriculture.

Scientific Sessions

These were held in the new Law School building, University City, with its capacious amphitheatres and meeting rooms, halls for exhibits, and with a restaurant, and many other roomy facilities. The general, section, and subsection meetings were held each day from Thursday, May 21 through Wednesday, May 27, with the exception of Sunday, May 24, when opportunity was provided for sightseeing tours and the traditional bullfights.

The presentation and discussion of main session and section papers were enhanced and expedited by simultaneous translation into the four official languages of the Congress: Spanish, English, French, and German.

A large number of short papers were also presented at subsection meetings but without simultaneous translation (*see* the JOURNAL, June 15, 1959, p. 573, for a list of program participants from the United States).

Closing Session

At the Closing Session on May 27, the Permanent Committee (which acts as an executive group for official Congress business) presented its recommendations on a number of matters to the general Congress assembly. Among items approved were the change of name of the I.V.C. to World Veterinary Congress and its parent organization to World Veterinary Association; a draft constitution of the W.V.A. was also approved, and a number of resolutions were accepted.

Merry; Dr. W. G. Magrane; Mrs. Merry; Dr. M. H. Jacobs; Mrs. B. M. Collins; Mrs. Jacobs; Dr. Collins; Dr. and Mrs. O. Norling-Christensen; Dr. C. R. Forman; Mrs. A. H. Quin; Mrs. Forman; Dr. Quin; Dr. K. M. Bullis; Dr. John Herrick; Mrs. Bullis; Dr. W. A. Hagan, chairman, U.S. Committee; Mrs. and Dr. M. W. Sullivan.

Also in the group but not in the picture were: Mrs. V. H. Miller, Dr. C. M. Christy, and Dr. R. E. Ruggles. Mrs. Magrane and Mrs. Ruggles followed on a later flight. Another tour group of 21 traveled by steamship, leaving New York May 9, on the Italian Line "Giulio Cesare." Other U.S. participants in the Congress traveled independently to Madrid. In all, some 200 persons from the U.S. were at the Congress.

Election of Officers

The following officers of the Permanent Committee of the World Veterinary Association were unanimously elected:

President—Prof. W. I. Beveridge, Australia.
Vice-President—Prof. Dr. Kurt Wagener, Germany.
Vice-President—Dr. W. A. Hagan, U.S.A.
Secretary-General and *Treasurer*—Prof. Dr. Jac. Jansen, Netherlands.
Deputy Secretary—Prof. Dr. L. de Blieck, Netherlands.

The resolutions adopted by this Congress, as well as action on the affiliation of a number of international associations of veterinary specialists with the World Veterinary Association, will be published in a future issue of the JOURNAL.

Dr. Scheidy Visits Israel, Spain, and England

During a recent five-week trip, Dr. Samuel F. Scheidy, president-elect of the AVMA, attended the XVIth International Veterinary Congress in Madrid, Spain, the Veterinary Symposium in Rishon Le Zion, Israel, and the annual meeting of the Royal College of Veterinary Surgeons in London, England.

Madrid

A participant in the XVIth International Veterinary Congress in Madrid (May 21-27), Dr. Scheidy served as chairman of the professional practice conference subsection. In addition, he delivered a paper in collaboration with Dr. J. E. Martin, of the University of Pennsylvania, on the pharmacology of certain phenothiazine ataraxias.

Israel

Prior to the Madrid conference, Dr. Scheidy attended the Veterinary Symposium in Rishon Le Zion, Israel, from May 10 to 17. The Symposium was sponsored by the Israel V.M.A. and the Israel Veterinary Institute, with the participation of the U. N.'s Food and Agriculture Organization.

The meeting was highlighted by the dedication of a new veterinary research laboratory, the first of its kind in Israel. Impressed with the tremendous strides Israel has made in basic research in animal production and disease control, Dr. Scheidy expressed the belief that "if Israel continues at its present pace, it will be a world leader in a few short years."

England

On June 2, in London, England, Dr. Scheidy also attended the annual meeting of the Royal College of Veterinary Surgeons. He was selected to represent the attendees in replying "to the toast of the guests." In his reply, Dr. Scheidy stressed the College's importance "to us in America" due to the numerous members of the organization that have participated in our teaching and research

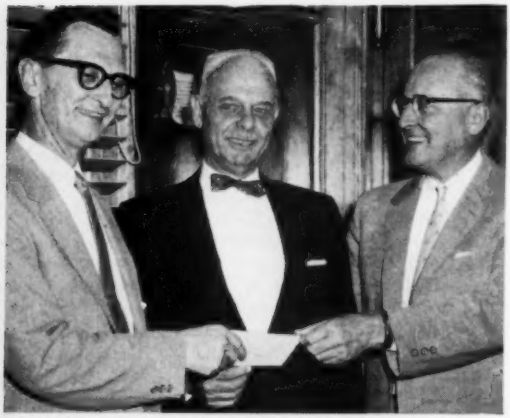
programs. He paid tribute to one of the College's Council members, Professor Beveridge of the University of Cambridge, "for the calm way in which he handled the difficult task of being chairman of an international body."

He concluded by saying how these purposes of the College are so "closely linked with the purposes for which our two great nations stand as leaders of the free world, for it is our duty, . . . to ensure that the under-developed nations of the world shall have their standards of living raised.

"The improvement of animal production," Dr. Scheidy continued, ". . . demands veterinary education and research not only in our own countries, but also in enabling the under-developed countries to have these benefits. . ."

N. A. F. V. Honors General McCallam

The National Association of Federal Veterinarians presented General J. A. McCallam (UP '17) with a check at a luncheon held in Washington, D.C., on May 7, 1959. The award was the Association's attempt to manifest its appreciation for the outstanding manner in which the General has represented the AVMA in Washington, and particularly, for his constant efforts to gain proper recognition for the veterinary profession.



Dr. Saulmon (left) is shown presenting The National Association of Federal Veterinarians' check to General J. A. McCallam (center) at a luncheon in Washington, D.C., May 7. Dr. Roy E. Willie, president of the Association, is at right.

Members of the Association in the Washington area attended the luncheon and Dr. E. E. Saulmon, chairman of the legislative committee, made the presentation to General McCallam. This action was authorized at the annual meeting of the Association in Miami Beach, Fla., last fall.

S/E. E. SAULMON, *Chairman.*

U. S. Chamber of Commerce Honors Midwest Feed Manufacturers' Association

The Midwest Feed Manufacturers' Association, in Kansas City, received a special award from the U.S. Chamber of Commerce during their forty-seventh annual meeting in Washington, D.C., last spring.

Left to right—Lloyd S. Larson, executive vice-president, Midwest Feed Manufacturers Association, Kansas City, Mo.; Frank Meuller, director of workshops, chamber of commerce of the United States, Washington, D.C.; Ray Ammon, director of feed division, The Quaker Oats Company, St. Joseph, Mo.

The award was presented to Mr. Ray Ammon, immediate past president of the association and director of the feed division, Quaker Oats Company, St. Joseph, Mo., for "initiating and carrying out a program to bring to practicing veterinarians the latest scientific findings in the field of animal and poultry nutrition research with the aim of improving and increasing the nation's food supply."

The veterinarians of the Iowa, Kansas, Missouri, and Nebraska Veterinary Medical Associations helped make the award possible through their cooperation and participation in the first annual Veterinary-Nutrition Conference held in Kansas City last December. The second annual Conference will be held this year at the President Hotel, in Kansas City, Mo., November 30 and December 1.

Dr. Helmboldt Succeeds Dr. Jungherr at the University of Connecticut

Dr. Charles F. Helmboldt has been appointed head of the Department of Animal Diseases in the College of Agriculture at the University of Connecticut.

His selection was announced May 23, 1959. Dr. Wayne N. Plastridge has been appointed as assistant department head. Dr. Helmboldt succeeded Dr. Erwin L. Jungherr, who retired June 30 (*see* the JOURNAL, June 15, 1959, p. 573).

Active in animal disease research, Dr. Helmboldt's studies have ranged widely over the fields of poultry and animal pathology. He has studied viruses in cattle, cattle nutrition, the histopathology

Dr. Charles F. Helmboldt

of bovine mastitis, and diseases of deer, swine, mink, and chinchillas. He joined the staff in 1946, and has done teaching, and extension service work, as well as research.

Dr. Helmboldt was born in Norwich, Conn., raised on a farm in Lisbon, Conn., and attended Norwich Free Academy. He received his bachelor's degree from the University of Connecticut in 1935, his D.V.M. degree from Michigan State University in 1938, and a Ph.D., in 1951, from the University of Connecticut.

Before entering the U. S. Army in 1942, he served as a junior veterinarian for the U. S. Department of Agriculture and conducted a private practice. Dr. Helmboldt recently spent six months on sabbatic leave in Germany, Switzerland, and Yugoslavia.

New School of Veterinary Science to Open at Purdue in Fall

The new School of Veterinary Science and Medicine at Purdue University will begin operation with the opening of the University in September. During the extended illness of Dean L. M. Hutchings (*see* p. 237), Dr. Edward O. Haelterman had been named assistant dean and professor of veterinary microbiology.

Part VI

Presidents of Constituent Associations

Alberta

Dr. Christopher H. Bigland
(ONT '41)

Illinois

Dr. Arthur K. Merriman
(IND '21)

Minnesota

Dr. Glen H. Nelson
(MIN '51)

AMONG THE STATES AND PROVINCES

Alabama

Upjohn Banquet Honors Two at API.—At the annual Upjohn Banquet for senior veterinary students at the Alabama Polytechnic Institute, Auburn, in February, Dr. J. L. Davidson presented awards to the outstanding student in each clinic (small and large animal) as determined by the votes of the faculty clinicians.

Recipients were: George Edward Pickens, Mayfield, Ky., for the Small Animal Clinic Award and Warren O. Nash, Jr., Georgetown, Ky., for the Large Animal Award (*see* Alabama Polytechnic Institute's commencement listing and picture on pages 246-247).

The awards consisted of a certificate, physician's tablet case, and a check for $50.

s/H. C. MORGAN, *Chairman, Publicity Committee.*

Colorado

College of Veterinary Medicine at Colorado State Gets New Electronic Equipment.—The Department of Veterinary Physiology at Colorado State University was recently equipped with modern electronic instruments designed to train students in the latest laboratory techniques.

The multi-channel recording instruments valued at $18,000 are capable of recording several physiological events simultaneously: blood pressure, pulse waves, heart sounds, respiratory rates, and amplitude, as well as velocity of respired air, muscle pull, nerve and muscle action potentials, body temperatures, and electrical activity of the heart and brain (electrocardiogram and electroencephalogram). Certain aspects of renal physiology, endocrinology, and metabolism can also be displayed using this equipment.

These versatile instruments, called physiographs, will be used by undergraduate students in Physiology and Pharmacology as well as for research. The instruments were developed at Baylor University's College of Medicine, at Houston, Texas, by the Department of Physiology and Biophysics through a grant-in-aid from the Heart Institute of the National Institutes of Health. Dr. Donald H. Will of the Colorado State University Department of Veterinary Physiology has received training at Baylor University on the operation of this electronic equipment.

s/DONALD H. WILL, *Correspondent.*

Kansas

Dr. Harwood and Williams Honored by Kansas State.—Two outstanding alumni of the School of Veterinary Medicine at Kansas State College, Drs. Nathan D. Harwood and Theodore S. Williams received Distinguished Service Awards at the annual banquet held during the twenty-first annual conference for veterinarians on June 1, 1959.

Dr. Nathan D. Harwood (KSC '18) is a former member of the School of Veterinary Medicine's faculty. In addition, he worked for many years in Kansas State's research laboratory producing hog cholera serum. Leaving the College's staff in 1926 to set up the Kansas Serum Company, he has engaged in the manufacture and sale of veterinary products ever since. Currently, Dr. Harwood is president of Patterson-Harwood Laboratories, the Manhattan Milling Company, and the Eureka Lake

A physiograph with accessories recently acquired by the Department of Veterinary Physiology at Colorado State University. The instrument was developed at Baylor University in Houston, Texas.

Dr. Nathan D. Harwood

Alfalfa Dehydrating Company. He is a past mayor of Manhattan, chairman of the Community Chest, and presently a member of the city commission.

After graduation, Dr. T. S. Williams (KSC '35) engaged in general practice in Kansas City, and Prairie View, Texas, for a year. From 1936 to 1945, he was employed by the former Bureau of Animal Industry in Des Moines, Iowa. He

Dr. Theodore S. Williams

became head of the Department of Pathology at Tuskegee Institute, Ala., in 1945. Two years later, Dr. Williams became dean of the School of Veterinary Medicine at Tuskegee. He is a member of Phi Kappa Phi, Gamma Sigma Delta, Beta Kappa Chi, and Alpha Phi Alpha, among many other organizations.

The Kansas State Conference for Veterinarians was held from May 31 through June 2.

Maine

State Association.—The annually scheduled spring meeting of the Maine V.M.A. was held at the Penobscot Valley Country Club in Orono on April 8, 1959.

The program included the following speakers and their respective subjects: Drs. F. Langdon Davis, Jr., Augusta—the practitioner's responsibilities in civil defense; R. S. Guthrie, New York State Veterinary College—milking machine defects; and R. W. Kirk, New York State Veterinary College—canine ear defects.

s/J. FRANKLIN WITTER,
Editor, The Maine Veterinarian.

Michigan

Southeastern V.M.A.'s Current Roster.—Elected to serve the Southeastern V.M.A. for 1959, are the following veterinarians: Drs. Charles Hodder, Detroit, president; Leonard Schreiber, Detroit, first vice-president; Gaylord MacCarty, Dearborn, second vice-president; Louis Rossoni, Dearborn, secretary; and Lyle Hartrick, Royal Oak, treasurer.

North Carolina

Eastern North Carolina Association Elects Officers.—At the last meeting of the Eastern North Carolina V.M.A. held at the Charcoal Steakhouse in Raleigh, on May 26, 1959, the following officers were elected: Drs. W. A. Potts, Mount Olive, president; C. B. Randall, Kinston, vice-president; and B. H. Brow, Goldsboro, was re-elected secretary-treasurer.

In addition, Dr. Hugh Simpson, Princeton, N.J., delivered an address on canine distemper and hepatitis.

s/BYRON H. BROW, *Secretary.*

Oklahoma

Dr. Pellette Retires from Federal Service.—Dr. Dudley B. Pellette retired as veterinarian in charge, at the Oklahoma Animal Disease Eradication Station, on Nov. 30, 1958, after 42 years of federal service.

Graduating from Kansas State College in 1912, Dr. Pellette was engaged in general practice in Kansas for five years, before he joined the Bureau of Animal Industry. For the following twenty years, he participated in the tick eradication program with assignments in Louisiana, Alabama, and Florida.

In 1936, Dr. Pellette was assigned to the

Dr. Dudley B. Pellette

Oklahoma City National Stockyards, Oklahoma City, as supervising veterinarian and after ten years was promoted to assistant veterinarian in charge in Oklahoma. During this assignment, he also served as secretary of the Oklahoma V.M.A. for one year. In 1951, Dr. Pellette was promoted to veterinarian in charge in Topeka, Kan., a position he held for over four years before returning to Oklahoma. Dr. F. W. Hansen, Jr., (see below) succeeds him.

Dr. and Mrs. Pellette are currently residing in Oklahoma City.

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Dr. Hansen Succeeds Dr. Pellette as Federal Veterinarian.—Dr. F. W. Hansen, Jr. (KSC '40) assumed the position of federal veterinarian in Oklahoma upon Dr. D. B. Pellette's retirement, Nov. 30, 1959.

Dr. F. W. Hansen, Jr.

After graduation, Dr. Hansen served for a year with the former Bureau of Animal Industry in Minnesota. He then entered private practice in Pelican Rapids, Minn., remaining there until 1943, when he joined the Army Veterinary Corps. In 1946, upon his discharge from the Army, Dr. Hansen resumed his practice in Pelican Rapids. In 1950, he accepted an appointment with the BAI and was stationed in Fort Morgan, Colo., for a time.

Dr. Hansen served as assistant veterinarian in charge in Vermont for a year before being transferred to Oklahoma as assistant veterinarian in charge in 1955.

Oregon

Dr. Derflinger Retires from Department of Agriculture.—Dr. E. R. Derflinger (OSU '15) retired in January after more than 42 years as assistant state veterinarian in the Oregon Department of Agriculture. He reached the maximum age for full-time state service, but is continuing limited part-time assignments in the

Dr. Earl R. Derflinger

department's disease control and meat inspection programs.

Dr. Derflinger was acting state veterinarian in Oregon during the California foot-and-mouth disease epizootic while the state veterinarian, the late Dr. W. H. Lytle, was making a first hand study of the disease in California.

His report on studies of and methods on intradermal immunization of cattle for anthrax was filed in the Congressional Library in 1938. This is based on work done in the Wood River Valley in Klamath County, Ore.

Prior to joining the state's agricultural department, Dr. Derflinger was engaged in private practice in Junction City, Ore., for a time, and then saw Army service in World War I.

A capacity crowd of over 5,000 heard the veterinary group sing their winning selection, "The March of the Men of Harlech," a fifteenth century Welsh song, at Washington State College's songfest, May 2.

Washington

Veterinary Choir at Washington State Wins College's Songfest.—For the fifth year in a row, the Veterinary Choir won the Men's Division of the annual Washington State College songfest, May 2, 1959.

The choir has made several appearances this year beside the songfest. The last performance of the 32-man group was on graduation day, when the choir's director, a graduating senior, Mark Keyes, was presented with an engraved plaque in appreciation for his splendid leadership over the last two years.

VETERINARY MILITARY SERVICE

Colonel Miller Makes Inspection Tour in Europe.—Colonel Robert R. Miller, V.C., U.S.A.F., assistant for veterinary services in the office of the Surgeon General, inspected Air Force veterinary services in Europe during May, 1959.

A member of the U. S. Committee of the International Veterinary Congress, Colonel Miller attended the convention in Madrid, Spain, May 21-27, and observed Air Force veterinary activities in Wiesbaden, Germany; London, England; and in Dublin, Ireland.

S/ROWLAND W. RUSHMORE,
Deputy for Professional Services.

Germany

Eighth Annual U.S.A.R.E.U.R.—U.S.A.F.E. Conference Held in Germany.—Eighty-eight Veterinary Corps officers of the U.S. Army and Air Force from 12 countries in Europe and the Middle East attended the eighth annual U.S. Army Europe—U.S. Air Force Europe Veterinary Conference held in Garmisch, Germany, May 6-8, 1959. This year, the Army Headquarters U.S.A.R.E.U.R., Heidelberg, Germany, sponsored the convention.

They discussed radiation hazards, particularly in radiological contamination of foods and water; also, the veterinarian's role in mass casualty care and on the preventive medicine team.

Opening addresses were given by Brig. Gen. Elmer W. Young (KSC '25), chief, veterinary division, office of the surgeon general, Washington, D.C., and Colonel Robert R. Miller (OSU '43), assistant for veterinary services, U.S.A.F., Washington, D.C.

While in Europe, General Young visited several of the veterinary units and investigated food inspection activities in Germany and France. He also represented the Surgeon General at the XVIth International Veterinary Congress in Madrid, May 21-27.

Panel discussions included: coordination of the U.S.A.R.E.U.R.—U.S.A.F.E.; and several on the various problems incurred in food inspection. Separate panels at the termination of the conference dealt with the specific problems peculiar to each of the services.

To the right are the leading personalities at the Veterinary Conference in Garmisch, Germany, May 6-8. Left to right—Colonel Curtis W. Betzold (COR '32), Medical Division, U.S.A.R.E.U.R.; Brig. General Elmer W. Young (KSC '25), chief of the veterinary division; Major General Alvin L. Gorby, chief surgeon, U.S.A.R.E.U.R.; Brig. General Joseph R. Ranck, quartermaster, U.S.A.R.E.U.R.; Colonel Marley C. Clark (MSC '35), U.S.A.F. E., Wiesbaden.

Graduating Class, 1959, School of Veterinary Medicine, Alabama Polytechnic Institute

Top row (left to right)—James R. Angel, Donald B. Applegate, David B. Aronson, Gary B. Beard, Paul D. Beard, Foster F. Beasley, Jr., Sidney D. Beckett, Burton Black.

Second row—John M. Black, Henry P. Brooks, Robert F. Chambless, Boyd C. Dodson, Ancel L. Duckworth, Jr., Arnold L. Fleisher, James R. Grace, James E. Gragg, Jr.

Third row—George W. Grimes, Garrett Hagan, Jr., James D. Hale, Jr., Robert D. Hawkins, James R. Holladay, Robert D. Horne.

Fourth row—Joe S. Larmon, John C. Lawrence, Rodney D. Lee, Alexander W. Macklin, Albert L. Martin, T. W. Megginson, Jr.

Fifth row—James W. Miller, Thomas F. Mink, James C. McLaughlin, Maurice C. McNay, Warren O. Nash, Jr., William P. Palmore.

Sixth row—George E. Pickens, William P. Pinson, William D. Roberson, Thomas E. Royal, Lawrence J. Scanlan, Nolan C. Sharp, Billy J. Smith, Paul C. Smith, Thomas C. Smyth.

Seventh row—Earl W. Stanton, Robert T. Sylvester, Patricia Anne Teer, Homer B. Tisdale, Jr., Howard G. Waite, Jr., Hilard E. Whitlock, Leslie R. Wilkinson, Alexander L. Williams.

Alabama Polytechnic Institute.—At the 1959 commencement exercises of the School of Veterinary Medicine, Alabama Polytechnic Institute, the following 51 candidates were presented for the D.V.M. degree:

James Robert Angel
Donald L. Applegate
David B. Aronson
Gary B. Beard
Paul D. Beard
Foster Farris Beasley
Sydney D. Beckett
Burton Black
John M. Black
Henry Palmer Brooks
Robert F. Chambless
Boyd C. Dodson
Ancel L. Duckworth, Jr.
Arnold L. Fleisher
James R. Grace
James E. Gragg
George W. Grimes
Garrett Hagan, Jr.
James David Hale, Jr.
Robert D. Hawkins
James R. Holladay
Robert D. Horne
Joe S. Larmon
John Clower Lawrence
Rodney D. Lee
Alexander W. Macklin
Albert L. Martin
Theodore W. Megginson
James W. Miller
Thomas Faye Mink
James C. McLaughlin
Maurice C. McNay
Warren O. Nash, Jr.
William Pearson Palmore
Alexander Lane Williams
George Edward Pickens
William Paul Pinson
William Douglas Roberson
Thomas Ervin Royal
Lawrence J. Scanlan
Nolan C. Sharp
Billy Jim Smith
Paul Clay Smith
Thomas C. Smyth
Earl William Stanton
Robert T. Sylvester
Patricia Anne Teer
Homer B. Tisdale, Jr.
Howard George Waite, Jr.
Hilard Elbert Whitlock
Leslie Ray Wilkinson

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Graduating Class, 1959, New York State Veterinary College, Cornell University

Cornell University.—At the 1959 commencement exercises of the School of Veterinary Medicine, Cornell University, the following 47 candidates were presented for the D.V.M. degree:

Ara Aris Ayanian
George Martin Baer
Alfred Lee Barkan
Albert Martin Beck
John Cornelius Burns
Harlow Jarvis Cameron
Edmond Alpheus Comans
Donald Richard Davidsen
Stanley Peter Duberman
Peter Edward Ebertz
Johan Robert Fineman
Charles Norman Gould
Richard Theodore Holmes
Wilbur Duane Kennedy
William Dale Knerr
Arthur Isiah Kronfeld
John Sidney Kyper
William Richard Latimer
Albert William Latschar
Roger Alan Laundy
Paul Christian Layer
Paul Rudolf Lindenmaier
John Raymond Long
John Edward Lowe
James George Mancuso
Alan Irving Marston
Richard Edward McCarthy
Eric Werner Mendel
Rodney Frederick Merz
John Charles Meyer
Frederick Augustus Murphy
Albert Wendell Nelson
Carl Daniel Nelson
Keith Henry Orts
Kenneth Meade Palmer
Paul Andrew Peterson
Sydney Bedford Kwatei Quartey
John Paul Rapp
George Nicholas Schatzle
Rudolph Schuster
Richard Edwin Shope, Jr.
George Clarence Shurtleff
Mary Elizabeth Van Order
Philip August Weber, Jr.
Andrew Jay Williamson
Albert Gene Wooding
William Emil Zitek

Graduating Class, 1959, College of Veterinary Medicine, Michigan State University

Top row (left to right)—Alan F. Berger, Roy C. Bergman, James C. Blakemore, Louis J. Blesch, Delwin K. Buckhold, Kermit L. Carlson, Carolyn S. Cartwright, Jacob Casper, Carl J. Christensen, J. Edward Clements, Roger A. Conant.
Second row—Henry B. Curtis, James B. Dalley, William J. Davis, Richard L. Dorner.
Third row—William J. Fedorko, Robert R. Filkins, James B. Fox, David O. Frey.
Fourth row—Edward F. Greene, Max S. Gritzmaker, Roderick C. Hartwick, Roger C. Hatch, Dean W. W. Armistead, Marvin C. Kaski, Thomas R. King, John W. Kinney, Ralph W. Levine.
Fifth row—Hilding L. Linderoth, Richard G. Menzel, John F. Mesman, Glenn K. Miller, Everett W. Parhiala, Charles T. Perkins, Kenneth E. Poole, John K. Reif, Dallas W. Rhoads, Robert E. Samson, Robert G. Sholtens.
Sixth row—Marjorie R. Smith, Dean S. Tribby, John M. Wagner, Kenneth A. Warner, Stanley D. Warner, Ramon J. Weber, Richard S. Webster, Nelson Westmoreland, Donald K. Weymouth, Philip K. Wolfe, William J. Wood.

Michigan State University.—At the 1959 commencement exercises of the College of Veterinary Medicine, Michigan State University, the following 49 candidates were presented for the D.V.M. degree:

Alan F. Berger
Roy T. Bergman
James C. Blakemore
Louis J. Blesch
Delwin K. Buckhold
Kermit L. Carlson
Carolyn S. Cartwright
Jacob Casper
Carl J. Christensen
J. Edward Clements
Roger A. Conant
Henry B. Curtis
James B. Dalley
William J. Davis
Richard L. Dorner
William J. Fedorko
Robert R. Filkins
James B. Fox
David O. Frey
Edward F. Greene
Max S. Gritzmaker
Roderick C. Hartwick
Roger C. Hatch
Marvin C. Kaski
Thomas R. King
John W. Kinney
Ralph W. Levine
Hilding L. Linderoth
Richard G. Menzel
John F. Mesman
Glenn K. Miller
Everett W. Parhiala
William J. Wood
Charles T. Perkins
Kenneth E. Poole
John K. Reif
Dallas W. Rhoads
Robert E. Samson
Robert G. Scholtens
Marjorie R. Smith
Dean S. Tribby
John M. Wagner
Kenneth A. Warner
Stanley D. Warner
Ramon J. Weber
Richard S. Webster
Nelson Westmoreland
Donald K. Weymouth
Philip K. Wolfe

Graduating Class, 1959, College of Veterinary Medicine, University of Minnesota

University of Minnesota.—At the 1959 commencement exercises of the College of Veterinary Medicine, University of Minnesota, the following 45 candidates were presented for the D.V.M. degree:

Frederick J. Alderink
Norbert E. Algiers
Carsten D. Anderson
Martin E. Bergeland
William N. Brascugli
James A. Burns
Raymond C. Callstrom
Richard E. Dierks
Charles W. Extrand
Charles J. Farho
Delmar R. Finco
Robert J. Fischer
Lloyd C. Foss
Peter H. Franz
Carlton C. Graves
John C. Gregg
Thomas J. Hagerty
Dale L. Haggard
Frank W. Hazard
Edward F. Jankus
Roland S. Jeans
David C. Johnson
Robert R. Jorgensen
James R. Juhl
James N. Kamp
John C. Landman
James A. Libby
George R. Nelson
Robert O. Nelson
Jerome E. Palet
Donald H. Person
Hans D. Peterson
Thayer E. Porter
Dennis P. Rahn
William P. Rogers
William T. Thompson
Alfred C. Trumble
John M. Turnbull
Lloyd VanPelt
George T. Walters
Thomas W. Wanous
Robert J. Weiner
Shannon C. Whipp
Thomas M. Willmus
Wilford A. Wohlin

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University of Montreal.—At the 1959 commencement exercises of the School of Veterinary Medicine, University of Montreal, the following 20 candidates were presented for the D.V.M. degree:

Jean-Paul Benoit
Viateur Bilodeau
Anicet R. Brault
Gilbert Yves Brodeur
Michel Calamel
Jacques Carrière
Bernard Chapuis
Gilles Charbonneau
Germain Gagnon
Jean Gosselin
Pierre Granier
Jules Harpin
Eugene M. Lareau
André Leclerc
Gilles Lussier
Jean-Paul Marcil
Claude Ménard
Michel P. Morésoli
Jean-Claude Panisset
Harold Slobod

(University of Montreal's commencement picture is on p. 250).

Graduating Class, 1959, School of Veterinary Medicine, University of Montreal

Top row (left to right)—Jean-Paul Benoit, Viateur Bilodeau, Anicet Brault, Gilbert Brodeur, Michel Calamel, Jacques Carrière, Bernard Chapuis.

Second row—Germain Gagnon, Gilles Charbonneau.

Third row—Jean Gosselin, Pierre Granier.

Fourth row—Jules Harpin, Eugene Lareau.

Fifth row—André Leclerc, Gilles Lussier, Jean-Paul Marcil, Claude Ménard, Michel Morésoli, Jean-Claude Panisset, Harold Slobod.

★ ★ ★ ★

University of Pennsylvania.—At the 1959 commencement exercises of the School of Veterinary Medicine, University of Pennsylvania, the following 40 candidates were presented for the D.V.M. degree:

H. L. Todd Addis
Jarvis J. Badgley
Norman K. Bailey
Uri Bargai
John C. W. Bauersfeld
Eugene A. Bodmer
Albert C. Brown
Robert I. Brown
Thomas J. Bucci
William H. Crawford, Jr.
David E. Davidson, Jr.
Robert J. Eberhart
John S. Evans
James M. Flickinger
Raymond W. Giuliani
George W. Heisey
Max J. Herman
Richard Hoey
George A. Keck
Leonard A. Kramar
William C. Lawrence
William C. Marlatt
Leigh A. Marsh
William L. Mullan, Jr.
Marvin A. Norcross, Jr.
H. Norman Pogust
Michael Ratner
Robert H. Reese
Conrad Richter
James M. Robinson
Thomas W. Ruffner
Bruce Schmucker
Edward Schwartz
John R. Seminatore
Richard W. Spertzel
Sheldon Steinberg
John B. Swinford
Daniel E. Vernon
Sherrill E. Ward
Paul W. Wetzel

Graduating Class, 1959, School of Veterinary Medicine, University of Pennsylvania

Top row (left to right)—James M. Robinson, Sherrill E. Ward, William H. Crawford, Jr., Paul W. Wetzel, Robert J. Eberhart, George W. Heisey, John S. Evans, David E. Davidson, Jr., Richard Hoey, Edward Schwartz, James M. Flickinger, George A. Keck, Raymond W. Giuliani, Bruce Schmucker.

Second row (left to right)—Richard O. Spertzel, Michael Ratner, Harry N. Pogust, Eugene A. Bodmer, Conrad B. Richter, Howard L. T. Addis, Thomas J. Bucci, Max J. Herman, Robert I. Brown, Albert C. Brown, Leigh A. Marsh, John C. Bauersfeld, John R. Seminatore.

Third row (left to right)—Robert H. Reese, William C. Lawrence, Sheldon Steinberg, John B. Swinford, Daniel F. Vernon, Jr., William C. Marlatt, William L. Mullan, Jr., Mrs. Thomas W. Ruffner*, Marvin A. Norcross, Jr., Jarvis J. Badgley, Norman K. Bailey, Uri Bargai, Leonard A. Kramar.

*Mrs. Thomas W. Ruffner stood in for her husband at graduation because Dr. Ruffner was ill.

DEATHS

Star indicates member of AVMA

★**Leo Anderson** (OSU '10), 70, Cedarville, Ohio, a large animal practitioner in Cedarville for nearly 50 years, died May 6, 1959.

Ira F. Dopp (STJ '20), 68, Craig, Mo., mayor of Craig, Mo., died May 18, 1959.

Born in Hannibal, Mo., Dr. Dopp moved to Craig in 1920. He had served the city as mayor several times since 1930.

John J. Emmons, 69, Ocala, Fla., formerly of Armstrong, Mo., died May 31, 1959.

A graduate of Kansas City Veterinary College, Dr. Emmons had been employed by the Federal Government as a meat inspector for 25 years. One of Dr. Emmons' children is also a veterinarian: Dr. E. E. Evans (OSU '40), of Decatur, Ill.

Fred C. Frye (KCV '14), Springfield, Mo., died Feb. 16, 1959, following an illness of several months.

Dr. Frye had engaged in general practice in Springfield for many years.

★**William S. Gochenour, Sr.,** (UP '13), 67, once a resident of Indianapolis, Ind., and former vice-president of Pitman-Moore Company, died on June 11, 1959, in a hospital at Daytona Beach, Fla. He had retired last year and moved to Deland, Fla.

Dr. William S. Gochenour

Born in York, Pa., in 1891, Dr. Gochenour received his preliminary education there and his V.M.D. degree from the University of Pennsylvania in 1913.

In 1915, he joined the Bureau of Animal Industry field inspection force and was engaged in foot-and-mouth disease eradication work in 1914, later being transferred to the Pathological Division in Washington. There, he was engaged in studies on shipping fever, anthrax, and other livestock diseases and their immunizing agents until 1937.

Dr. Gochenour became associated with Pitman-moore in 1938 as director of their biological laboratories and in 1952 was made vice-president in charge of veterinary relations. During World War II, he was active in allied joint research on bacteriological warfare.

Surviving are two sons: Lt. Colonel W. S. Gochenour, Jr. (UP '37), Silver Springs, Md., and Dr. Raymond B. Gochenour (UP '38), Kensington, Md.

James H. Hartzog, 85, Denmark, S. Car., died April 13, 1959, following an illness of several weeks.

He was the oldest licensed veterinarian in the county, remaining in practice and farming as long as his health permitted.

Earl H. Hedrick (OSU '13), 69, Bainbridge, Ohio, died April 18, 1959. Dr. Hedrick was a veteran of World War I and practiced in Bainbridge for 46 years.

Greydon S. Hicks (GWU '12), 67, Fresno, Calif., died in a local hospital on April 21, 1959.

Born in Brighton, Mich., Dr. Hicks served seven years on the Ford estate in Milford, Mich., in the early 20's. While there, he cared for the automotive tycoon's private dairy herd, riding horses, and for the sheep that were used to provide wool for his automobiles.

Dr. Hicks was with the U.S.D.A. in Fresno, working in the animal research division. He retired from that position six months ago and was with the Sierra Valley Livestock Company at the time of his death.

Charles Prothro Hill (API '43), 38, Newnan, Ga., died in the Veterans Hospital in Atlanta, on May 5, 1959.

Dr. Hill practiced at various times during his career in La Grange, Griffin, and Newnan and was assistant professor of small animal medicine at the University of Georgia. He is a past president of the Georgia V.M.A.

Joseph Golden Irons (UP '11), 76, Nephi, Utah, died April 15, 1959, of a cerebral hemorrhage.

Dr. Irons was active in the civic affairs of Nephi and was instrumental in the establishment of a National Guard unit there. In recent years, he served with the U. S. D. A. as the city's meat inspector. He was still actively engaged in practice at the time of his death.

George B. Kramschuster (MCK '17), 64, Bloomer, Wis., died suddenly April 12, 1959. After graduation, he practiced in New Auburn for one year before moving to Bloomer.

Benjamin C. Ornbaum (IND '08), 77, Petersburg, Ind., died in an Evansville, Ind., hospital on Jan. 16, 1959.

After graduation in 1908, he practiced in Brownsburg and Plainfield, moving to Petersburg in 1909. Dr. Ornbaum was honored recently by the Southwestern Indiana V.M.A. upon completion of his fiftieth year in the profession.

★**Robert Miles Sarde** (UP '13), 67, Smyrna, Del., died May 20, 1959.

A first lieutenant in the regular Army from 1917 to 1919, Dr. Sarde received five battle stars. During World War II, he served as a major and then a lieutenant colonel from 1941 to 1946, receiving two battle stars and an Army Commendation Ribbon.

Dr. Sarde was director of livestock sanitation for the Delaware State Board of Agriculture from 1923 to 1939.

★**Wilson Mitchell Smotherman** (SW '14), 69, Huntsville, Texas, died May 21, 1959, after a short illness.

Born in Tennessee, Dr. Smotherman had spent most of his life in Texas, and had lived in Huntsville for 32 years. While in practice there, he had served as veterinarian for the Texas prison system until several years ago. He had also worked for the Texas Livestock Sanitary Commission for 11 years, and was once assistant state veterinarian.

★**Alfred Isaac Spurr** (AVC '88), 93, Mount Washington, Mass., died at the Fairview Hospital in Great Barrington on May 5, 1959. He was one of the oldest veterinary graduates in America.

✦ ✦ ✦

Other Deaths Reported.—The following deaths have been reported. The usual information for an obituary was not supplied.

John Ayer (CIN '18), Cincinnati, Ohio, died in January, 1959.

Carl M. Case (IND '16), 81, Sumner, Ill., died May 31, 1959.

Charles Jackson Grissom, 67, Mantachie, Miss., died May 18, 1959.

C. V. Lard, 67, Glenarm, Ill., died March 18, 1959.

J. O. Martin, 85, Green City, Mo., died May 26, 1959.

W. A. Meyers (CIN '12), Tiffin, Ohio, died Feb. 19, 1959.

Horace Grier Roland, 87, Dexter, Ga., died April 29, 1959.

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*Belloff, G. B.: Calif. Vet. 9:16 (Nov.-Dec.) 1955.

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What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and radiographs depicting a diagnostic problem are usually published in each issue.

History.—Three German Shepherd Dog bitches, approximately 1½ years old, were examined radiographically for evidence of hip dysplasia. The radiographs (fig. 1A, B, C) were taken with the dogs anesthetized and in a ventrodorsal position, the hindlimbs extended posteriorly in sagittal planes.

Make your diagnosis from the pictures—then turn the page ▶

Here Is the Diagnosis

(Continued from preceding page)

Diagnoses.—1A—Dysplasia of both acetabulums, coxa magna of the right femur, and osteoarthritis (probably traumatic) of the right hip joint.

1B—Normal for the breed.*

1C—Normal with a questionable "taint" in the left acetabulum.

Several veterinarians experienced in diagnosing hip dysplasia were asked to comment on these films. All were in agreement on the diagnosis of 1A. Clinically, this dog showed a flat "rump," a wide spread between the trochanters, a limited range of motion, and tenderness of the right hip joint. On 1B and 1C, all examiners "hedged." Some commented that the acetabulums appeared large and shallow, especially on the left side of 1C. All examiners agreed that it is difficult to find more desirable joints in this breed. (In the interest of establishing more uniformity in diagnoses, readers are asked to comment on these films and submit others that may aid in standardizing opinion.)

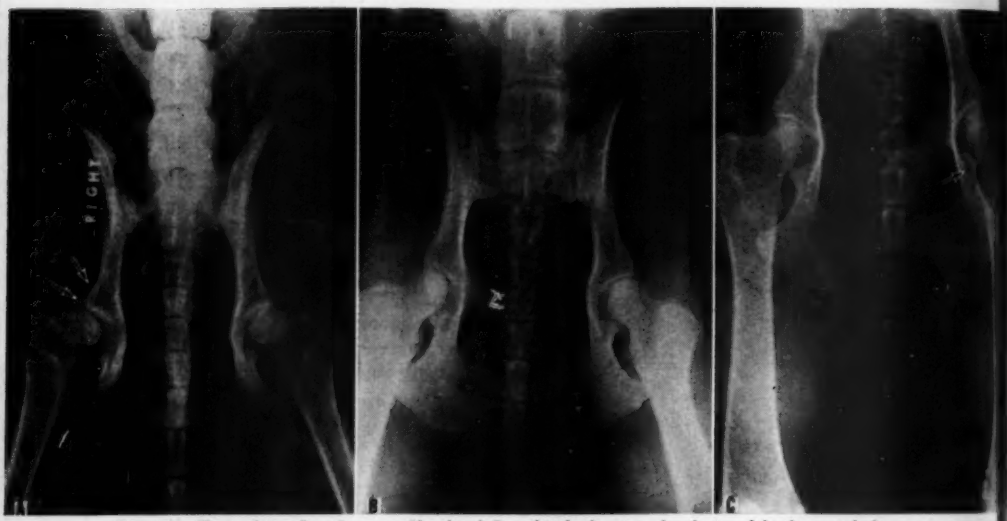

Fig. 1A—The pelvis of a German Shepherd Dog bitch showing dysplasia of both acetabulums, coxa magna of the right femur, and osteoarthritis (probably traumatic) of the right hip joint (arrows).

Fig. 1B—Pelvis of a German Shepherd Dog bitch considered to be normal.

Fig. 1C—Pelvis of a German Shepherd Dog bitch considered to be normal with a questionable "taint" in the left acetabulum (arrow).

*Opinion of several experienced observers who viewed the radiographs.

Comment.—Hip dysplasia, a faulty development of the hip joint, is attracting increasingly more attention among veterinarians and breeders. Most of the knowledge on the subject relates to diagnosis or genetics. A thorough study of the anatomic development of both the normal and abnormal hip joint is needed to give a better understanding of the disease.

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REFERENCES: 1. Pollock, S.: Control of Nonspecific and Specific Enteritis in the Dog; read at the First Regional Conference on the Nitrofurans in Veterinary Medicine, Wilmington, Delaware (June 5, 1958). 2. Fisher, G. W.: Oral Use of Furadex in Canine Diarrhea, N. A. Vet. 39:133-134 (Mar. 15) 1958.

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There is a growing demand for radiological examination of the hips of prospective breeding stock, and the veterinarian is expected to give an accurate estimation of the anatomic normality of the hips of each dog examined. This calls for better technique in taking, developing, and reading of radiographs.

Excerpts from two recent observations are quoted:

> If the hereditary factors are dominant, then the eradication of the disease would be possible by bold and somewhat ruthless methods, as no animals "carrying" the genetic factor responsible but not themselves exhibiting the disease, could escape unnoticed radiographically. On the other hand, if the genetic factor concerned is a simple recessive, the creation of the "carrier" animal would hinder eradication considerably. From preliminary observations it seems that the genetic factors concerned may not be "simple" but associated with a combination of factors in which case the position from this point of view may prove to be complex.[1]

The other report states:

> Dysplasia of the hips of dogs is significantly increased by purposeful breeding of dysplastic parents. From observations made from my German Shepherd Dog kennel, it is shown that: (1) Where both parents were dysplastic, the disease was transmitted in diagnosable form to 87 per cent of the offspring. The population limits of the incidence were 63.6 to 97.7 per cent. (2) Where one parent (the female) was dysplastic and one (the male) was normal, the disease appeared in diagnosable form in 62.4 per cent of the offspring. (3) Where parents both had normal hips, the disease was present in diagnosable form in 17.0 per cent of the offspring, and the population limits of the incidence were 2.6 to 59 per cent.[2]

This report was submitted by the Riser Animal Hospital, Skokie, Ill.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

References

[1]Singleton, W. B.: Hip Dysplasia in the Dog. Proc. British Small Anim. Vet. Assoc., First Ann. Congress (1958): 75-80.

[2]Snavely, J. G.: The Genetic Aspects of Hip Dysplasia in Dogs. North Ohio V.M.A., Toledo, March 24-25, 1959.

Instructions to Authors

JOURNAL *of the AVMA*

Exclusive Publication.—Articles submitted for publication are accepted with the understanding that they are not submitted to other journals, which is ethical publication procedure.

Manuscripts.—Manuscripts, including **footnotes, references,** and **tables,** must be typewritten, **double-spaced,** on 8½- by 11-in. bond paper, and the original and one carbon copy, submitted. One-inch margins should be allowed on the sides, with 2 in. at top and bottom. Articles should be concise. Short, simple sentences are clearer and more forceful than long, complex ones.

Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good contrast for reproduction and must be large enough to stand reduction, if necessary.

Drawings, graphs, and charts should be made clearly and accurately in India ink on white paper and a glossy print of them submitted when possible. Numbers or letters appearing on graphs or charts should be large enough to allow for any reduction necessary for the chart or graph to fit JOURNAL pages. Blue lines in graph paper drop out in reproduction; therefore, if lines are required they must be drawn in black ink. All illustrations should bear the name of the author and the illustration number on the back.

Tables.—Tables should be simple and typed **double space.** Complex tables are not conducive to perusal. It is wiser to summarize complex material than to tabulate it.

References.—References should be typed **double space,** in alphabetical order, and should be prepared in the following style: name of author, title of article, name of periodical with volume, year, and page numbers. References to journals not commonly known should give the complete name of the periodical, and where published so that they may be added to our reference files. When books are cited, the name of publisher, location, edition, and year should be given.

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1. Harris, J.R., and Clarkson, T.B., Prevention of Relapses in Milk Fever, Vet. Medicine, *12*:696 (Dec. 1955)

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AVMA Represented at Senate Committee Hearing

Dr. J. O. Knowles, practitioner from Miami, Fla., member of the AVMA House of Delegates, and chairman of its Advisory Committee, testified July 15, 1959, before the Committee on Finance of the U.S. Senate with respect to the Self-Employed Individuals' Retirement Act of 1959. Dr. Knowles' statement follows:

I appear today as the representative of the American Veterinary Medical Association to support S. 1979, introduced by Senator Smathers, and H.R. 10 and H.R. 9, sponsored respectively by Representative Keogh and Representative Simpson (R., Pa.), bills to encourage the establishment of voluntary pension plans by individuals. Our Association has for many years endorsed the principle of legislation of this type.

Both measures under consideration today, are designed to provide tax deferment for the self-employed individual, whereby that person may each year set aside a limited portion of his own income for investment in certain types of retirement annuity, or a specific retirement trust. These are restricted voluntary plans, with certain transactions prohibited, and penalty provisions are provided also, an example of the latter being withdrawal of amounts before age 65.

A problem common to numerous individuals is to provide a source of income for themselves and their families in later years related to the standard of living established during the more productive years. A solution, though difficult under the present high tax rate and inflated costs, would be in the interest of the self-employed person and the national economy. The Congress has recognized the objective as merited by amendments to the Internal Revenue Code which provides for qualified pension plans for corporate employees, including the executives. Employees participating in these approved plans do not, under the law, have to include the employer's contribution as gross income until pensions are received, and contributions by the company are deductible in the year made.

Why this tax relief to one group in our economy —the corporate employee? There are many doctors of veterinary medicine in the latter. Why are those engaged in the private practice of their profession —the self-employed, denied by law the tax relief offered to corporations and their employees? It is an existing discrimination which we think is unfair and unsound. The American Veterinary Medical Association believes the practicing veterinarian and other self-employed persons should be encouraged and assisted to provide their own funds for their old age and retirement, by enactment of the bills providing for tax deferment.

The veterinarian starts his professional career after six to eight years in preprofessional and professional study. He is almost twenty-six years of age when entering practice, and starts to earn income. Then he has to build his practice. He is left with a relatively few years of top earning before his income begins to diminish. The obvious funds for providing for retirement should come from the surplus during these years of highest return. The income tax now siphons off much of this surplus. We have this problem in common with other self-employed groups and professional people. However, we also have a specific problem that should be brought to your attention. The nature of our work requires that in addition to the conventional expenses, such as family, home, and so forth, we must provide a building in which to practice. A veterinary hospital is a single purpose building and as such is a poor investment. It is of no use to a widow and is of little use to anyone besides another veterinarian, so although it represents a considerable investment of the family's funds, it has a limited market and does not provide comparable security against disability, demise, or for retirement. A veterinary hospital will cost from a minimum of perhaps $15,000 to as much as $200,000.

There has been considerable discussion before this committee that H.R. 10 is class legislation and is intended for the people in the upper income tax brackets. I am able to speak for a group who are vitally interested in persuading you to act favorably on this legislation, but are certainly not in the upper income tax group. The average net income for a veterinarian in private practice is approximately $10,000 per year. This income, of course, is calculated on a lifetime basis, which means that there are many years of income much less than that before he builds up to his peak, a few years of income considerably more, and then several years of income that again is reduced. The structure of the income tax is such that much of his excess is siphoned off during his peak years. To earn his $10,000 a year, he works an average of 60 hours a week, and must invest from that income whatever is required to provide the facilities for his practice.

Considering all these factors, both professional and individual, the self-employed veterinarian has little opportunity under the present tax law to take advantage of the more prosperous years of his practice in order to provide economic security for his family and himself by embarking on a sound retirement program.

There seems to be concern that this law would enable a self-employed individual to provide for himself but not for his staff. I wonder if this is realistic. The tremendous increase in the number of retirement programs in this country shows that the principle is being so well received it certainly will find its way into most businesses. An employer, self-employed or not, must compete in the general labor market for his staff, and he must provide adequate inducements in order to obtain competent employees. If most of the employed people of the country have retirement plans, our employees will too. For example, our hospital in Miami is not under the wage and hour law, and yet the law profoundly influences our pay scale. Years ago

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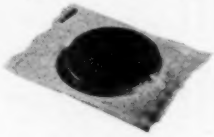

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when it was first enacted, it brought about a raise in our pay scale equal to that required by the wage and hour law. We have, since that time, paid equal to or above the scale, and as economic necessity we shall continue that policy.

The veterinarian is a prime example of the self-reliance so vital to the building of this country. We are anxious to stand on our own feet, to be responsible for our own actions and, as much as possible, solve our own problems, including that of our old age. The American Veterinary Medical Association urges your committee to permit us this privilege by acting favorably on H.R. 10 and S. 1979.

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Grade GS-7 Eliminated for Veterinarians

The U.S. Civil Service Commission has approved the Department of Agriculture's request to raise the entrance grade for veterinarians without previous experience to GS-9. AVMA influence is reflected in this favorable action, which enables newly graduated veterinarians to qualify for positions in the federal service at an entrance salary of $6,135 per annum, the second step of the grade GS-9 salary scale. The entrance grade and salary for such veterinarians who have not had a year or more of professional veterinary experience following graduation have heretofore been grade GS-7, at $5,430 per annum.

In announcing the action taken by the Civil Service Commission, the Agricultural Research Service emphasized that it has numerous openings throughout the United States in both meat inspection and livestock disease control and eradication activities. All vacancies are being filled at the grade GS-9 level, $6,135 per annum. Opportunities for advancement to positions of higher responsibility are good. Veterinarians who are interested in employment are encouraged to contact the Personnel Division of the Agricultural Research Service, U.S. Department of Agriculture, Washington 25, D.C.; or they may contact the nearest Inspector in Charge of federal meat inspection, the Federal Veterinarian in Charge of livestock disease control and eradication work, or officials in charge of other veterinary activities of the Agricultural Research Service.

The Agricultural Marketing Service has approximately 50 vacancies for veterinarians in full-time poultry inspection at a similar grade level and salary. Inquiries should be addressed to any AMS area personnel office or to the Veterinarian in Charge, Veterinary Poultry Inspection offices, at Philadelphia, Atlanta, Chicago, Des Moines, Dallas, or San Francisco.

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Seal and Minute Book of the U.S.V.M.A.

1866

The annual meeting of 1866 was held at the New York College of Veterinary Surgeons, of which Alexandre Liautard was the nominal head. Many of the later meetings were held here, or at the American Veterinary College after Dr. Liautard became its head in 1875. Also suggestive of the considerable influence he had in Association affairs is his election as its first secretary, later three times as president, his editorship of the *American Veterinary Review*, and the fact that he held one or more official positions almost without exception up to the year 1900.

At this meeting, a committee was appointed to investigate charges against Robert Jennings "for tampering with the records and withholding other papers belonging to the Association." Upon their report, Dr. Jennings was expelled from the Association. Four pages were thereupon removed from the minute book, and the following notation was made: "Margin of leaves on which Dr. Jennings inserted minutes of meetings held in Philadelphia previous to the formation of this Association and while acting as secretary."

From the writings of both Dr. Jennings and his son in the 1880's, it is apparent that his version of the events leading to the founding of the U.S.V.M.A. did not correspond with the official record. As indicated in the notation above, the Association considered the minutes inserted by Jennings to be of meetings (A.V.A.) *previous to the formation of this Association*. Thus there would seem to be no question but what the founding fathers considered the U.S.V.M.A. an entirely new organization.

It is also evident that Jennings was disgruntled over not having been elected the first president of the Association. According to his son, he withdrew his nomination in the interests of party harmony on the alleged promise of the post the following year, but was again passed over by a coalition of New York and Massachusetts members.

Whether this is true or not is a matter of conjecture, but it is perhaps significant that these two states dominated the Association for two decades. It is also apparent that a number of the Pennsylvania delegates withdrew from the organization meeting.

At the 1866 meeting, Dr. C. H. Curtis was elected president; and Drs. Burden and Thayer were re-elected secretary and treasurer.

✦ ✦ ✦

CHARLES M. WOOD, V.S., M.D., third president of the U.S.V.M.A., came to this country from England in 1835 and started working as a blacksmith, but soon took up veterinary practice. During the 1850's, he was associated with George Dadd in the Boston Veterinary Institute, and was a regular contributor to Dadd's short-lived *American Veterinary Journal* and to the *Veterinarian* (London).

Writing on "Veterinary Medical Knowledge" in 1858, he deplores the apathy "so generally manifested, by the *certified* Veterinary Surgeons of this country, in regard to the diffusion of veterinary knowledge . . . . they claim the right to remain silent on the subject, unless in the stable, or bar-room . . . . If the truth is not told, people will suppose that error is truth; and go on inflicting misery, instead of ministering relief."

Wood appears to have been the brains behind the Boston Veterinary Institute; he was alone in insisting upon high academic standards. In 1861, he was employed as a civilian inspector of animals for the Army of the Potomac. An entirely self-educated practitioner, he had an office in a livery stable in Boston on what was later the site of the Parker House.

While he was characterized as "proficient in the details of the profession. . . ." he was also characterized as "brusque, antagonistic, and as aggressive as one could be."

Obviously, enough of the others thought sufficiently well of Dr. Wood to successively elect him censor, treasurer, president, and censor three times again before his death in 1869. His M.D. degree would appear to have been self-acquired.

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In small-animal urinary tract infections, FURADANTIN swiftly achieves high bactericidal concentrations in the urine, and in a high percentage of cases eliminates incontinence, dysuria, frequency, and straining. Of 32 dogs and cats recently treated,[1] 29 showed rapid clinical improvement. Often, there is marked improvement by the 4th day and complete recovery in 7 to 14 days.[2]

In canine tracheobronchitis, FURADANTIN given for 5 days stopped the coughing in 95% of 75 cases; in some dogs, complete symptomatic relief was gained in 48 hours.[3]

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REFERENCES: 1. Mosier, J. E., and Coles, E. H.: Vet. Med. 53:649 (Dec.) 1958. 2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

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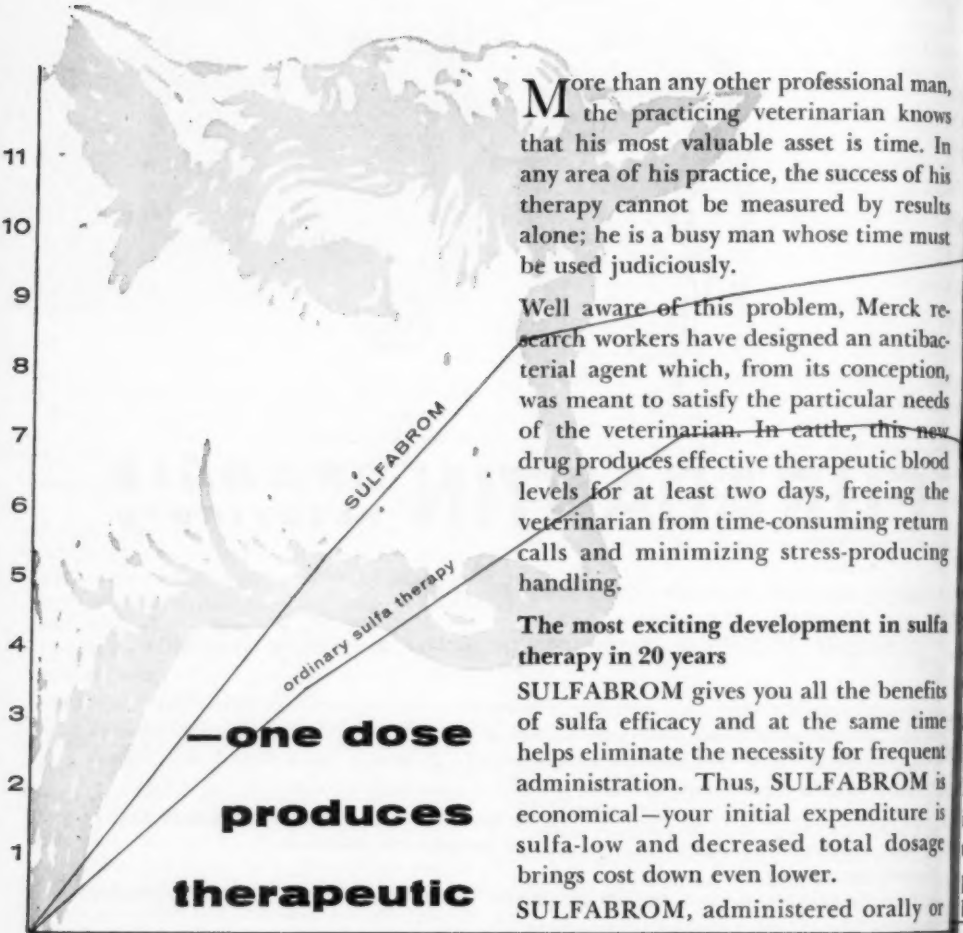

More than any other professional man, the practicing veterinarian knows that his most valuable asset is time. In any area of his practice, the success of his therapy cannot be measured by results alone; he is a busy man whose time must be used judiciously.

Well aware of this problem, Merck research workers have designed an antibacterial agent which, from its conception, was meant to satisfy the particular needs of the veterinarian. In cattle, this new drug produces effective therapeutic blood levels for at least two days, freeing the veterinarian from time-consuming return calls and minimizing stress-producing handling.

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SULFABROM, administered orally or

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intraperitoneally to cattle, produces effective blood levels lasting up to 48 hours—frequently long enough to eliminate *any* repetition of dosage.

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Although SULFABROM is notable for producing effective levels in rapid time, once it has entered the blood stream its speed of action slows down considerably. SULFABROM is excreted very slowly; this accounts for its long-lasting effect. In cattle, detectable amounts may be present in the urine for as long as six days. Blood levels remain high, sometimes for as long as 53 to 60 hours. And, because it is excreted so slowly, seldom is the amount of SULFABROM passing through the urinary tract ever large enough to cause crystalluria.

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acute septic mastitis "uneventful recovery";
metritis "back on feed and eating normally" in two days;
foot rot "in 48 hours the cow was able to stand and started to eat";
pneumonia "in five days' time this calf was back to normal";[1]
as well as *scours*, winter dysentery, coccidiosis, shipping fever, listerellosis and miscellaneous infectious conditions, such as peritonitis and infected wounds.

SULFABROM—single-dose sulfa therapy available exclusively to veterinarians

In sum, SULFABROM represents the very latest advance in sulfa therapy. Effective against a full range of infectious diseases, economical to use by any standards, SULFABROM is your answer to the pressing problem of repeat calls and handling time in the treatment of almost any infection.

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SULFABROM Buffered Powder
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COMING MEETINGS

Notices of coming meetings must be received 30 days before date of publication.

American Association of Veterinary Bacteriologists. Annual meeting. Division of Veterinary Medicine, Iowa State College, Ames, Iowa, Aug. 22, 1959. C. H. Cunningham, Michigan State University, College of Veterinary Medicine, East Lansing, secretary.

Ninety-Sixth Annual Meeting, American Veterinary Medical Association, and Third Pan American Congress of Veterinary Medicine. Joint meeting. Kansas City, Mo., Aug. 23-27, 1959. H. E. Kingman, Jr., executive-secretary, AVMA, 600 S. Michigan Ave., Chicago 5, Ill. B. D. Blood, secretary-general, Directing Council, Pan American Congress of Veterinary Medicine, P.O. Box 99, Azul, F.C.N.G.R., Argentina, S.A.

Washington State Veterinary Medical Association. Annual meeting. Desert Inn, Richland, Aug. 31 to Sept. 1, 1959. Mr. Robert M. Ford, 2406 Boyer Ave., Seattle 2, Wash., executive secretary.

Colorado Veterinary Medical Association. Annuel convention. Shirley Savoy Hotel, Denver, Colo., Sept. 3-4, 1959. Gail H. Gilbert, 5500 Wadsworth Ave., Arvada, executive secretary.

West Virginia University. Symposium in magnesium and agriculture. West Virginia University, Medical Center Auditorium, Morgantown, W. Va., Sept. 3-4, 1959. D. J. Horvath, program committee chairman.

Electron Microscope Society of America. Seventeenth annual meeting. Ohio State University, Columbus, Sept. 9-12, 1959. Sydney S. Breese, Jr., Plum Island Animal Disease Laboratory, Greenport, L.I., N.Y., program chairman.

New York State Veterinary Medical Society. Annual meeting. Grossinger's, Grossinger, N.Y., Sept. 16-18, 1959. F. H. Fox, chairman.

South Dakota Veterinary Medical Association. Annual meeting. Sheraton-Johnson Hotel, Rapid City, S. Dak., Sept. 17-18, 1959. Glenn E. Duncan, Tyndall, secretary.

New Mexico Veterinary Medical Association. Annual meeting, Western Skies Hotel, Albuquerque, N. M., Sept. 21-22, 1959. E. R. Leslie, 907 Alamosa, Carlsbad, N.M., secretary.

Pennsylvania State Veterinary Medical Association. Annual meeting, (Combined with the University of Pennsylvania School of Veterinary Medicine's Seventy-Fifth Anniversary celebration). Bellevue-Stratford Hotel, Philadelphia, Pa., Oct. 1-3, 1959. R. C. Snyder, Walnut St. and Copley Rd., Upper Darby, Pa., secretary.

Eastern Iowa Veterinary Association, Inc. Forty-sixth annual meeting. Roosevelt Hotel, Cedar Rapids, Oct. 15-16, 1959. C. B. Thayer, Medical College, State University of Iowa, Iowa City, Iowa.

Interstate Veterinary Medical Association. Annual meeting. Sioux City, Iowa, Oct. 22-23, 1959. Dr. Don Rubel, 3209 38th St., Sioux City, secretary.

Animal Care Panel. Annual Convention. Sheraton Park Hotel, Washington, D.C., Oct. 29-31, 1959. William I. Gay, Animal Care Panel, 2101 Constitution Ave., Washington 25, D.C., publicity committee chairman.

Southern and West Virginia Veterinary Medical Associations. Combined meeting. Lord Baltimore Hotel, Baltimore, Md., Nov. 1-4, 1959. A. A. Husman, P. O. Box 91, Raleigh, N. Car., secretary, Southern V.M.A., and H. J. Fallon, 200 5th St., W. Huntington, W. Va., secretary, West Virginia V.M.A.

Missouri, University of. Thirty-fifth annual veterinary conference. University of Missouri, School of Veterinary Medicine, Columbia, Nov. 2-3, 1959. Cecil Elder, chairman.

Foreign Meetings

International Association of Veterinary Food Hygenists. Second Symposium. Basel, Switzerland, May 15-21, 1960. Dr. A. Clarenburg, 1, Sterrenbos, Utrecht, The Netherlands, president.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Medical Association, the first Thursday of each month. James L. Chambers, 4307 Normanbridge Rd., Montgomery, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. Dan P. Griswold, Jr., 714 S. 39th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. Cecil S. Yarbrough, 4121 U.S. 90 West, Mobile, Ala., secretary.

Northeast Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ALASKA—Anchorage Group of the Alaska V. M. A., the last Wednesday of each month at Fort Richardson Officers' Club or Thompson's Restaurant 6th and I Streets, Anchorage, Alas. Lt. Colonel E. H. Akins, Surgeon's Office, U.S.A.R.A.L., Fort Richardson, Alas., secretary to the Alaska V. M. A.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. J. W. Langley, Jr., P.O. Box 5013, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. Gwyn Chapin, 2215 E. Calle Vista, Tuscon, Ariz., secretary.

ARKANSAS—Pulaski County Veterinary Medical Society, the second Tuesday of each month. Harvie R. Ellis, 54 Belmont Drive, Little Rock, Ark., secretary-treasurer.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. John S. Blackard, 420 Appian Way, Richmond, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16th St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Paul S. Chaffee, 2333 McKinley Ave., Fresno, Calif., secretary.

Humboldt-Del Norte Counties Veterinary Medical Association, the second Tuesday of January, May, September, and November. Dr. M. Lunstra, P. O. Box 734, Eureka, Calif., secretary-treasurer.

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Thursday evening of the month. James L. Frederickson, 17 Nile St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of each month. William P. Matulich, P. O. Box 121, San Luis Obispo, Calif., secretary-treasurer.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. V. Todorovic, 47 Mann Ave., Watsonville, Calif., secretary.

Northern California Association of Veterinarians, the second Tuesday of the month. Andrew F. Giambroni, P.O. Box 782, Red Bluff, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. T. J. Carleton, 325 W. Lockeford St., Lodi, Calif., secretary-treasurer.

Orange Belt Veterinary Medical Association, the second Monday of each month. R. Y. Foos, P.O. Box 955, Victorville, Calif., secretary-treasurer.

Orange County Veterinary Medical Association, the third Thursday of each month. H. M. Stanton, 1122 S.E. U.S. Highway 101, Tustin, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of the month. R. M. Grandfield, 416 Stephens Rd., San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of the month. R. R. Rediske, 833 Vallejo Ave., Novato, Calif., secretary-treasurer.

Sacramento Valley Veterinary Medical Association, the second Wednesday of the month. E. C. Story, 4819 "V" St., Sacramento 17, Calif., secretary-treasurer.

San Diego County Veterinary Medical Association, the fourth Tuesday of the month. Robert F. Burns, 7572 North Ave., Lemon Grove, Calif., secretary-treasurer.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Barbara G. Shirley, Canoga Park, Calif., secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, Calif., secretary.

Santa Barbara-Ventura Counties Veterinary Medical Association, every three months, no set date. Gerald M. Clark, 5415 8th St., Carpinteria, Calif., secretary-treasurer.

Santa Clara Valley Veterinary Medical Association, the last Tuesday of the month. Robert L. King, 1269 Grant St., Santa Clara, Calif., secretary-treasurer.

Southern California Veterinary Medical Association, the third Wednesday of the month. Mr. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

COLORADO—Denver Area Veterinary Medical Society, the fourth Tuesday of every month. Gene M. Bierhaus, 2896 S. Federal Blvd., Englewood, Colo., secretary-treasurer.

Northern Colorado Veterinary Medical Society, the first Wednesday of each month, in Fort Collins. Dr. James Voss, Veterinary Hospital, Colorado State University, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Medical Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. A. P. Mayer, Jr., R.F.D. 2, Newark, Del., secretary-treasurer.

DISTRICT OF COLUMBIA—District of Columbia Veterinary Medical Association, the second Tuesday evenings of January, March, May, and October. R. B. Gochenour, 10109 Ashwood Dr., Kensington, Md., secretary-treasurer.

FLORIDA—Big Bend Veterinary Medical Association, meets the first Sunday of each month at 5:00 p. m., at the Tallahassee Dining Room, Tallahassee. C. Paul Vicken, P.O. Box 309, Tallahassee, secretary.

Central Florida Veterinary Medical Association, the first Friday of each month at 8:00 p. m., place specified monthly. L. R. Poe, 753 W. Fairbanks Ave., Winter Park, Fla., secretary-treasurer.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. Fred Jones, 3606 S. Dale Mabry, Tampa, Fla., secretary.

Hillsborough County Veterinary Medical Society, the second Monday evening of each month. For additional information as to the location of each meeting, contact: J. J. Metz, Jr., 5207 Nebraska Ave., Tampa 3, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodson's Restaurant, Stephen C. Hite, 5807 105th St., Jacksonville 10, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. John Webb, P.O. Box 183, Cantonment, Fla., secretary-treasurer.

Palm Beach Veterinary Society, the last Thursday evening of each month. McArthur Dairy Building, Four Points, W. Palm Beach. B. W. Bigger, 2833 S. 4th St., Fort Pierce, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. John S. Haromy, Route #1, Box 107-A, Lake Wales, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Joe B. O'Quinn, 1690 E. 4th, Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. G. L. Burch, P.O. Box 405, Ocala, Fla., secretary-treasurer.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. Robert E. Cope, 127 E. Mason, Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Medical Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Clare L. Bromley, 634 Northside Dr., N.W., Atlanta, Ga., secretary.

Georgia-Carolina Veterinary Medical Association, the second Monday of each month at 8:00 p.m., at the Town Tavern, Augusta, Ga. J. A. Schmitz, 1711 Gwinnett St., Augusta, Ga., secretary.

North Georgia Veterinary Medical Association, quarterly, no set date, the spring meeting at the Veterinary School, Athens, Ga. S. J. Shirley, Commerce, Ga., secretary.

Southeast Georgia Veterinary Medical Association, quarterly, date and meeting place varies. Hugh F. Arundel, P.O. Box 153, Statesboro, Ga., secretary.

South Georgia Veterinary Medical Association, the second Sunday of each quarter at 3:30 p.m., at the Radium Springs Hotel, Albany, Ga. M. W. Hale, Route 2, Tifton, Ga., secretary.

ILLINOIS—Central Illinois Veterinary Medical Association, June 9, Sept. 9, and Dec. 10, 1959. Paul B. Doby, 4 Owens Lane, Springfield, secretary.

Chicago Veterinary Medical Association, the second Tuesday of each month, Charles H. Armstrong, 1821 Davis St., Evanston, secretary.

INDIANA—Calumet Area Veterinary Medical Association, the first Thursday of each month. Bruce Sharp, Box 166, Hobart, Ind., secretary-treasurer.

Central Indiana Veterinary Medical Association, the

second Wednesday of each month. P. T. Parker, 224 N. Mill St., Plainfield, Ind., secretary-treasurer.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. Stanton Williamson, 217 W. Chippewa St., South Bend, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotton, P.O. Box 183, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month except June, July, and August at 6:30 p.m., Breeze House, Ankeny, Iowa. S. L. Hendricks, secretary-treasurer .

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, 202 S. Hazel Ave., Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Veterinary Medical Society, the Second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. T. F. Bartley, P.O. Box 454, Cedar Rapids, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, P.O. Box 162, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, 202 S. Stone St., Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. W. A. Danker, Dows, Iowa, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. R. H. Folsom, P.O. Box 323, Danville, Ky., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

LOUISIANA—New Orleans Veterinary Medical Association, the third Thursday of every month at the Monteleone Hotel, New Orleans, at 8:30 p. m. Ronald C. Francis, 6421 Chef Menteur Highway, New Orleans, La., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Leonard D. Krinsky, 6111 Hartford Rd., Baltimore, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Jerry Fries, 2070 E. Main St., Owosso, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert W. Acton, 4110 Spring Rd., Jackson, Mich.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. Alvin R. Conquest, P.O. Box 514, Grand Blanc, Mich., secretary.

Southeastern Michigan Veterinary Medical Association, the fourth Wednesday of every month, September through May. Louis J. Rossoni, 24531 Princeton Ave., Dearborn 8, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kan., secretary.

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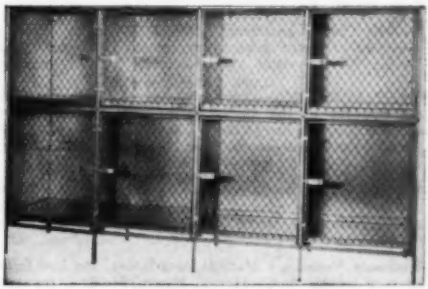

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Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, R.D. 1, Box 284A, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April, except December, at the Irvington House, 925 Springfield Ave., Irvington, N.J. Bernard M. Weiner, 787 Clinton Ave., Newark, N.J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. James R. Tanzola, Upper Saddle River, N.J., secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. L. Smith, P.O. Box 938, Trenton, N.J., secretary.

South New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collmont Diner, Collingswood, N.J. Marvin Rothman, 718 Dwight Ave., Collingswood, N.J., secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, the third Wednesday of each month, Fez Club, Albuquerque. Donald W. Fitzgerald, 1825 Lomas Blvd., N.E., Albuquerque, N.M., secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. C. G. Sims, 2450 Battleground Ave., Greensboro, N. Car., secretary.

Eastern North Caroline Veterinary Medical Association, the last Tuesday evening of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary-treasurer.

Piedmont Veterinary Medical Association, the last Friday of each month. J. G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. Vita Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, 451 E. Galbraith Rd., Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, 3120 Valley View Dr., Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, 8208 Carnegie Ave., Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, 6941 Far Hills Ave., Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. C. Gale, Wooster, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the Fourth Tuesday of each month, at 9:00 p.m. Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, 1683 W. Bancroft St., Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, 42 W. Market St., Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Claude A. Tigert, 3032 N.W. 68th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month at the City-County Health Building, 4616 E. 15th St., Tulsa, Okla. Arlen D. Hill, 5302 E. 11th St., Tulsa, Okla., secretary.

Tulsa Association of Small Animal Veterinarians, first and third Mondays, City-County Health Dept. T. E. Messler, 3104 E. 51st St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m. Ireland's Restaurant, Lloyds, 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Robert J. Mallorie, P.O. Box 155, Silverton, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the Uni-

(*Continued on adv. p. 53*)

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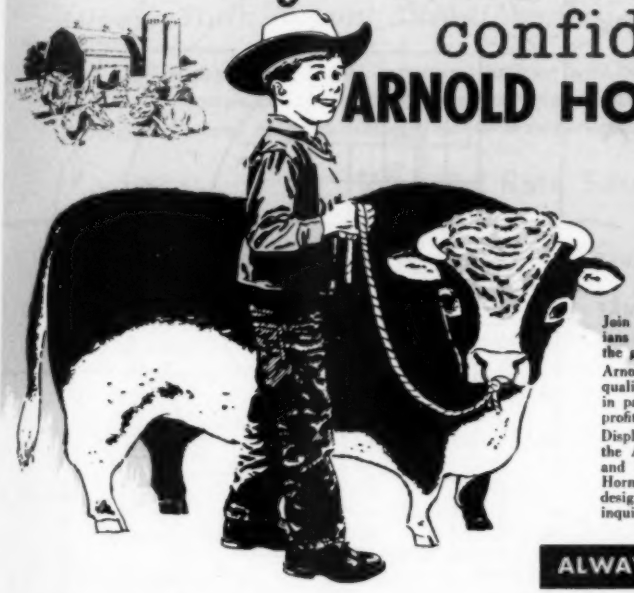

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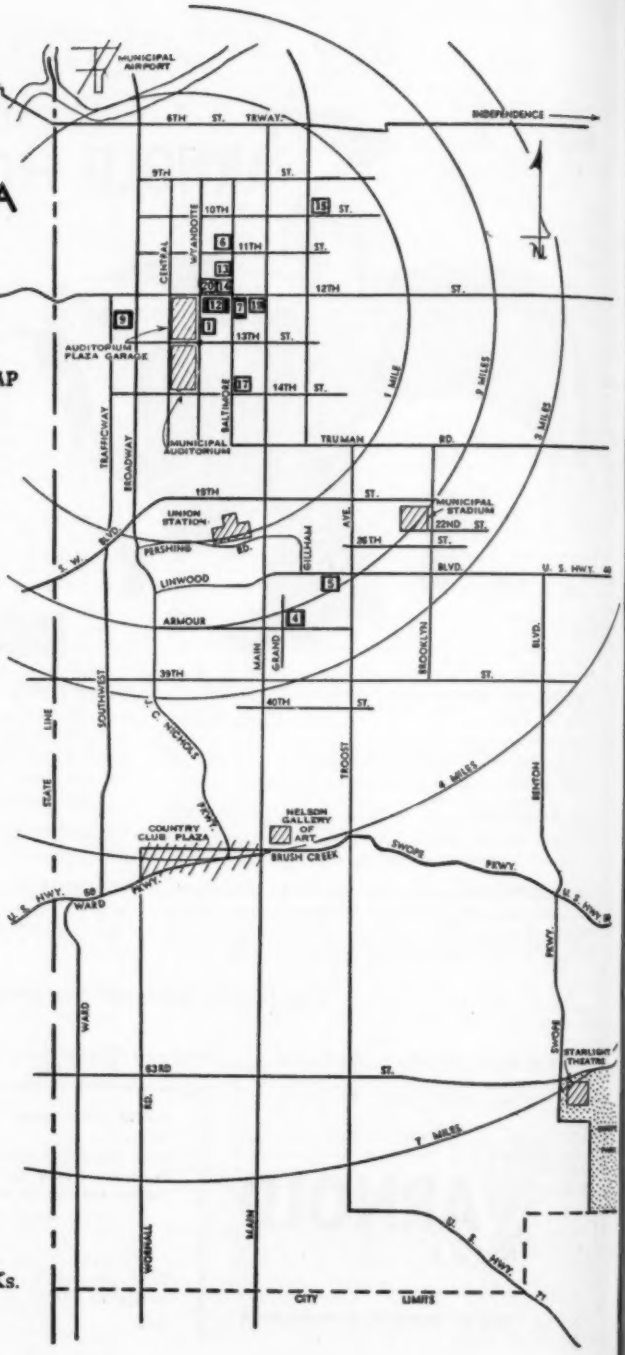
Map of the
GREATER KANSAS CITY AREA

ALL HOTELS NOT IN WALKING DISTANCE OF MUNICIPAL AUDITORIUM ARE WITHIN 10 MINUTES BY PUBLIC TRANSPORTATION

HOTEL LIST AND DOWNTOWN MAP

NO.	HOTEL
1	Aladdin, 1213 Wyandotte
4	Bellerive, 214 East Armour Blvd.
5	Berkshire, 1021 E. Linwood
6	Continental, 11th & Baltimore
7	Dixon, 12th & Baltimore
9	Kansas Citian, 1216 Broadway
12	Muehlebach, 12th & Baltimore
13	New Yorker, 1114 Baltimore
14	Phillips, 12th & Baltimore
15	Pickwick, 10th & McGee
17	President, 14th & Baltimore
19	Senator, 17 W. 12th St.
20	State, 12th & Wyandotte
21	Town House, 7th and State, Kansas City, Ks.



HOTEL INFORMATION — KANSAS CITY, MO., CONVENTION

Ninety-Sixth Annual AVMA Meeting, Aug. 23-27, 1959

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Committee on Local Arrangements. The Bureau will clear all requests and confirm reservations.

Hotel and Rate Schedule

Map No.	Hotel	Single	Double	Twin	Suite
1.	Aladdin*	$4.50-8.50	$ 6.50-10.50	$ 9.50-12.00	$17.00-30.00
4.	Bellerive*	5.00-9.00	8.00-12.00	9.00-13.00	From $18.00
5.	Berkshire*	5.00-7.00	7.00-10.00	8.50-10.00	From $14.00
6.	Continental*	6.50-11.00	8.50-13.50	10.00-14.00	$20.00-32.00
7.	Dixon	4.50-7.00	6.50-9.00	8.00-12.00	- - - - - - -
9.	Kansas Citian	3.50-8.00	5.50-11.00	7.00-14.00	From $10.00
12.	Muehlebach*	Headquarters Hotel — No Room Accommodations			
13.	New Yorker	5.50-12.00	8.00-14.00	9.50-14.00	$23.00
14.	Phillips*	7.50-10.50	9.50-13.00	11.50-14.00	$20.50-35.00
15.	Pickwick*	5.85-10.85	6.35-10.85	8.35-12.50	From $14.00
17.	President*	6.50-10.00	9.50-13.00	11.00-15.00	$25.00
19.	Senator	3.50-7.00	5.00-10.00	6.00-10.00	$15.00
20.	State	4.75-6.50	7.50-8.75	8.75-9.25	- - - - - - -
21.	Town House*	5.50-12.00	10.00-13.50	11.00-16.00	From $23.00

*100 per cent air-conditioned; in other hotels listed, majority of rooms air-conditioned.

FAMILY PLAN—The above hotels offer a "family plan" whereby children under 14 years of age will be accommodated in the same room with their parents at no extra charge. If more than one room is required to accommodate children, the hotel will charge only the single rate for each room.

MOTELS—Reservations for motels in the Kansas City area may be made through the Kansas City Convention and Visitors Bureau, 1030 Baltimore Ave., Third Floor, Kansas City 5, Missouri.

PLEASE USE APPLICATION ON REVERSE SIDE FOR HOTEL ACCOMMODATIONS

Application for Hotel Accommodations

1959 AVMA Convention — Kansas City, Missouri

The Convention and Visitors Bureau will make every effort to place you according to your expressed wishes, or to best advantage elsewhere if that is not possible and you desire us to do so.

Please give us the complete information requested below. At least four choices of hotels, or more if you desire, are necessary. Arrange for double occupancy of rooms wherever possible; only a limited number of single rooms is available.

Date.....

Please make hotel reservation in accordance with the following:
Accommodations desired:

Hotel.....First Choice

Hotel.....Second Choice

Hotel.....Third Choice

Hotel.....Fourth Choice

.....Room WITH bath for one person Rate per room desired $..... to $.....

.....Room WITH bath for two persons (double bed) Rate per room desired $..... to $.....

.....Room WITH bath for two persons (twin beds) Rate per room desired $..... to $.....

.....Large room WITH bath for..... persons Rate per room desired $..... to $.....

.....Suite—Parlor, ...Bedroom(s) with bath for ..... persons. Rate per suite desired $.....

Arrival date....., hour.....A. M. .....P. M.
Departure date.....

If reservation cannot be made in one of the hotels indicated shall we place you elsewhere? Yes..... No.....

Please check your mode of transportation: Car..... Train..... Plane..... Bus.....

Rooms will be occupied by (NAMES OF ALL PARTIES MUST BE LISTED)
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NAME	STREET ADDRESS	CITY	STATE

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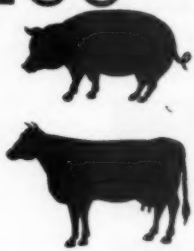

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PACKAGING AND COMPOSITION—5 pound canister—125 Grams. Each pound of Terramix A-B-D-25 contains 25 grams of oxytetracycline activity, plus 2,000,000 units of Vitamin A, 200,000 units of Vitamin D_3, 2 grams of Riboflavin, 4 mg. of Vitamin B_{12}, with expeller soybean oil meal as the diluent, Q.S.

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versity of Pennsylvania School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

Georgia-Carolina Veterinary Medical Association—see GEORGIA.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. Jack E. Habluetzel, Route 1, Box 65-N, Ingleside, Texas, secretary.

Dallas County Veterinary Medical Association, the first Tuesday of each month at 7:30 p.m., at a place to be specified. Frank N. Black, 12830 Preston Rd., Dallas, Texas, corresponding secretary.

UTAH—Salt Lake Small Animal Hospital Association, the first Monday of every month, at the Holiday Inn, 3040 South State St., Salt Lake City, at 12:15 p. m. Douglas H. McKelvie, 1220 S. State St., Salt Lake City, Utah, secretary-treasurer.

VIRGINIA—Central Virginia Veterinary Association, the second Thursday of each month at 8:00 p. m., except July and August, at a place in Richmond to be announced monthly. Edwin M. Crawford, secretary-treasurer.

Northern Virginia Veterinary Conference Association, the second Tuesday of each month. T. P. Koudelka, P.O. Box 694, Harrisonburg, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwestern Virginia Veterinary Medical Association, the first Thursday of each month. D. F. Watson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle. Roy C. Toole, 10415 Main St., Bellevue, secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. B. D. Benedictson, 3712 Plummer St., Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the third Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.) C. R. Carlson, 1109 E. LaSalle Ave., Barron, Wis., secretary.

Coulee Region Veterinary Medical Association, the third Wednesday of every other month. F. N. Petersen, Box 127, Cashton, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. Raymond Pahle, 10827 W. Oklahoma Ave., Milwaukee, Wis.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. L. C. Allenstein, 209 S. Taft St., Whitewater, Wis., secretary.

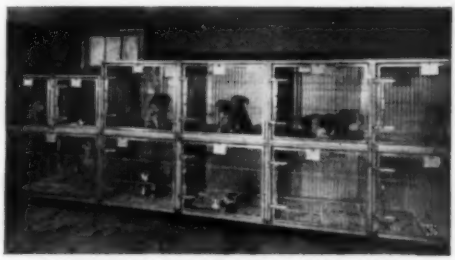
Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. John B. Fleming, 209 E. 4th St., Marshfield, Wis., secretary.

New Paint Absorbs Sound

Of interest to veterinarians with hospitals located in well-populated areas, a new paint has been developed which reportedly helps to reduce the noise level in a building.

Tests indicated that noise in rooms painted with the new product was 25 to 33 per cent less than in rooms painted conventionally. The reason for this is attributed to the porosity and softness of the paint film. The manufacturer claims that one coat provides effective sound conditioning, although a second coat may be added for additional soundproofing, and that sound control is still effective after a period of two years.—*Dog World (June, 1959): 6.*



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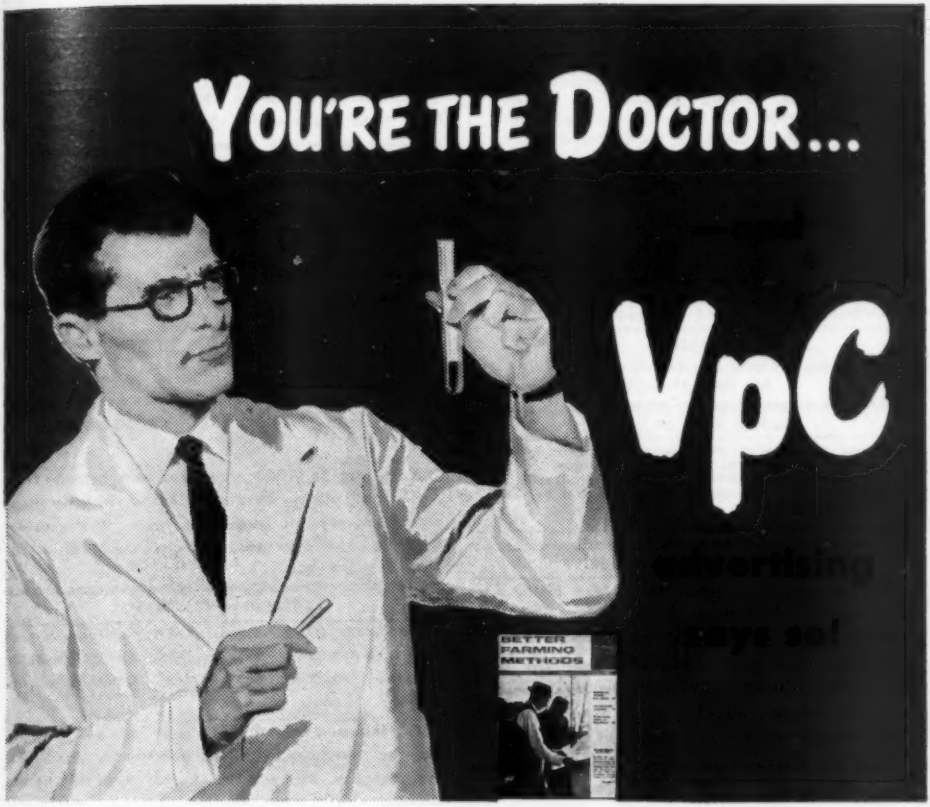

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Wanted—assistant veterinarian for small animal hospital in Chicago. Good opportunity. Address Box L 33, JOURNAL of the AVMA.

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Progressive recent graduate wanted to assist two practitioners. Liberal salary. Must have Missouri or reciprocal license. Address Box M 29, JOURNAL of the AVMA.

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Associate veterinarian for modern small animal hospital near Philadelphia. Mature person with experience and good reference desired. Starting salary $7,500 per annum. Address Box M 36, JOURNAL of the AVMA.

Excellent opportunity—veterinarian to assist in well-established practice, percentage equine. Florida license required. Address Box M 37, JOURNAL of the AVMA.

German veterinarian, 35, unmarried, wants a long time assistantship in the States. Good experience, especially in small animal practice. Address Box M 45, JOURNAL of the AVMA.

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References: 1. Vigue, R. F.: J.A.V.M.A. 133:326 (Sept. 15) 1958. 2. Shaw, J. C.: Personal communication. 3. Pollock, S.: Vet. Med. 54:97 (Feb.) 1959. 4. Rabin, P. H.: Personal communication. 5. Hoffer, S. H.: Personal communication. 6. Weir, H. T., and Hazelrig, J. W.: Personal communication. 7. Beck, J. W.: Personal communication. 8. Bull, W. S.: Personal communication. 9. Fessenden, P. E.: Personal communication. 10. Lohmeyer, C.: Personal communication.

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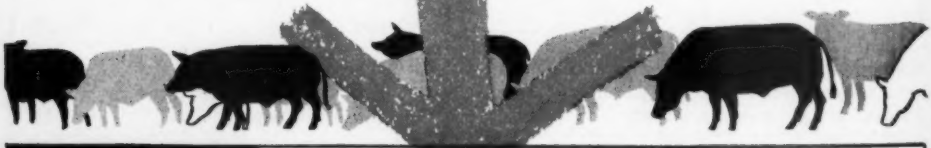

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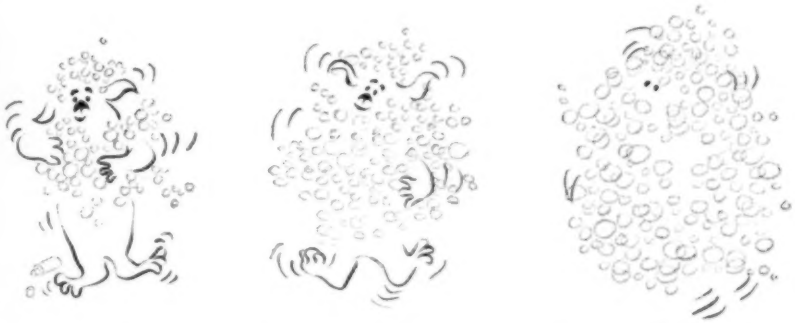

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